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INTRACAVITARY (MONALDI) SUCTION

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INTRACAVITARY suction (Monaldi) has been employed at the Edward Sanatorium for the past three years. During this time it has come to occupy a definite and important place in the treatment of pulmonary tuberculosis. The early hope that it would prove an easy way of closing cavities and would therefore supplant some of the older forms of therapy has not been fulfilled. The difficulty in establishing it in small cavities, its uncertainty, and the long period which it must be continued have limited its use to those patients who, because of low vital capacity, cannot be treated by pneumothorax, thoracoplasty, or extrapleural collapse. In these rather common cases in which no other form of treatment is possible it offers a possible means of arresting the disease. It is also used occasionally to reduce the size of extremely large cavities so the thoracoplasty will be more likely to close them.

Because the method is relatively new and is not widely used it seems advisable to describe the technique in detail and to present a brief summary of each case in which it has been used.

THE TECHNIQUE

When the procedure was first used it was assumed that if no free pleural space could be found with the pneumothorax needle at the site chosen for drainage, adhesions were present and the trocar could be inserted safely. In two of the early cases this assumption proved to be incorrect and the catheter was inserted through a free pleura. A fatal empyema with bronchial fistula resulted. Since that time the operation has been performed in two or three stages.

Read at the Twenty-fifth Annual Meeting of the American Association for Thoracic Surgery, Chicago, Ill., May 5 and 6, 1944.

Editor's Note: Apologies are due to Dr. Head and the various discussers for the long delay in publishing this paper and the discussions. The paper was part of a symposium of which the leading paper was by Dr. H. McLeod Riggins on "Studies of the Pathogenesis, Dynamics and Closure of Tension Cavities." Repeated efforts have been made in vain to obtain Dr. Riggins' manuscript. Finally, it has been decided to publish Dr. Head's paper with the discussions of the symposium. Dr. Arthur Vineberg's presentation was a motion picture without a manuscript.

At the first stage the pleura is explored through a small intercostal incision, if the catheter is to be inserted from in front, or by resection of a small piece of rib, if the catheter is to be inserted from the back. If adhesions are found the wound is sutured and the catheter inserted one or two weeks later. A small amount of lipiodol or a small metal clip is left at the level of the pleura so that on subsequent x-rays the relation of the proposed site of entrance to the cavity can be determined. If a free pleura is encountered the pleura is opened and a small iodoform gauze pack is inserted just to the level of the pleura. A metal clip is attached to this for the purpose of location by x-ray and the wound is sutured. Ten days or two weeks later the pack is removed and the presence of adhesions verified. The wound is again sutured and one week later the catheter is inserted. Until fairly recently the catheter was inserted at the time of removal of the pack and was brought out through a small stab wound just below the incision. So commonly this resulted in infection and opening of the incision that the third stage was adopted.

The cavity is located first on both anteroposterior and lateral x-rays. Most apical cavities are best approached from in front through the first interspace. Small ones located posteriorly can be entered most easily from behind. Cavities located elsewhere than in the apex are approached directly at the point where they are nearest to the chest wall.

Placing of the catheter requires extreme care. The cavity is located first by exploratory aspiration with a long 19 gauge needle. This is attached to a 20 c.c. syringe. The point of the needle can be assumed to be in the cavity when air or pus can be freely aspirated. The depth of the cavity is then noted and marked on the cannula which is the type used in draining the gall bladder and of a size to admit a No. 14 French catheter. The trocar and cannula are then inserted in the determined direction to the determined depth and the catheter put in place. This is at first put in farther than is necessary and then, when the cannula has been withdrawn, is pulled out until a mark, so placed on it that the tip will be about one inch within the cavity, appears at the skin.

It is extremely important that the tube be fixed firmly in place by an air-tight adhesive dressing and that great care be taken that it is not pulled out, but that the mark on the catheter be kept always at the level of the skin.

Continuous suction is maintained through a bottle trap by a small electric pump (Stedman). This is kept up for from six to ten months. The catheter is then left in place and open for another month. If the cavity reopens, suction is again applied. If it remains closed, the tube is removed.

RESULTS

During the past three years 29 patients have been treated by this method. Of these, 16 are still living and 13 are dead. For the most part the procedure was used as a last resort in desperate cases in which the patient had too extensive involvement and too little vital capacity to permit of any other form of collapse therapy—a group in which a fatal outcome seemed almost certain and any recoveries could be classed as pure gain.

The causes of death in the 13 patients who died were as listed in Table I.

The extent of cavity closure in those who died was as shown in Table II.

TABLE I. CAUSES OF DEATH

CAUSES	NUMBER
Empyema with bronchial fistula	2
Hemorrhage from the treated cavity with extensive spill	1
Tuberculous meningitis	1
Normal progression of extensive soft tuberculosis	5
Tuberculous enteritis and laryngitis	3
Cardiac decompensation	1

TABLE II. EXTENT OF CAVITY CLOSURE

EXTENT	NUMBER
Closed	3
Greatly decreased in size	7
Unaffected	3

TABLE III. RESULTS IN LIVING PATIENTS

RESULT	NUMBER
Cavity closed; sputum negative	7
Treated cavity closed; patient greatly improved	6
Cavity reopened after thoracoplasty	1
Cavity decreased in size, hemorrhage, worse	1
Unimproved with poor prognosis	1

In the 16 patients who are alive the results are as shown in Table III.

Of the 29 patients, 22 had too extensive disease and too little vital capacity to permit of any other form of treatment. Without direct treatment of the cavity the prognosis was hopeless.

The results in these 22 cases are as shown in Table IV.

TABLE IV. RESULTS IN TWENTY-TWO PATIENTS WITH EXTENSIVE DISEASE

RESULTS	NUMBER
Well	5
Greatly improved	6
Greatly improved; died of cardiac failure	1
Died of progression of tuberculosis in other lung areas	4
Died of enteritis and laryngitis	2
Died as a result of treatment	2
Alive; worse	1
Alive; unimproved	1
Total	22

In 7 of the early cases the procedure was used in patients who might have been treated otherwise. The results in these are as shown in Table V.

At first there seemed reason to hope that in patients with extensive soft tuberculosis, closure of the cavities would be followed by healing of the un-

TABLE V. RESULTS IN SEVEN EARLY CASES

RESULTS	NUMBER
Well	2
Cavity reopened after thoracoplasty	1
Died as result of treatment	2
Died of progression of tuberculosis	1
Died of tuberculous enteritis	1

excavated lesions. This hope has not been fulfilled. In nearly every instance the cavity closed or became smaller but the other lesions progressed, and after stopping the suction the cavities reopened and enlarged.

CONCLUSIONS

From the foregoing recounted experience the following conclusions are suggested:

1. Intracavitary suction should never be used when any other form of treatment is applicable.

2. It is indicated for quiescent cavities of relatively long standing which are not surrounded by soft active disease in patients in whom low vital capacity precludes the use of other measures.

3. It is of value in reducing the size of extremely large excavations as a preliminary to thoracoplasty.

DISCUSSION ON "STUDIES OF THE PATHOGENESIS, DYNAMICS AND CLOSURE OF TENSION CAVITIES" BY DR. MCLEOD RIGGINS; "THE DETERMINATION AND TREATMENT OF PRESSURE CAVITIES IN PULMONARY TUBERCULOSIS" BY DR. ARTHUR VINEBERG; AND "INTRACAVITARY (MONALDI) SUCTION" BY DR. JEROME R. HEAD

DR. JOHN ALEXANDER, Ann Arbor, Mich.—Dr. Riggins and Dr. Gearhart have added important and much-needed fundamental information about the still somewhat obscure matter of tension cavities. The application of this information to the therapeutic management of our patients is especially difficult because of the many variable factors that are involved. For example, the dramatically successful treatment reported by Dr. Riggins, in which a cavity closed immediately upon the aspiration of air, and has remained closed for a year, is unlikely to be duplicated often in view of the fact that prolonged aspiration through Monaldi drainage tubes so rarely completely and permanently closes a cavity.

It seems likely that the effectiveness of collapse therapy in closing tension cavities depends largely upon the degree to which the strictured bronchus resists the expelling of air and secretions from the cavity during expiration or coughing, or during the collapsing effect of whatever operation is used. A "positive pressure threshold" of 15 or 20 cm. of water may become changed by collapsing operations to a higher or a lower value as a result of a change in the position of, and tension upon, the stenotic bronchus that is responsible for tension within the cavity. If this assumption is true, a preoperative determination of the positive pressure threshold in a given case would not necessarily be a constant measure of the resistance of the stricture to the discharge of the air or fluid contents from a cavity following pulmonary collapse and, therefore, would not necessarily be a measure of the likelihood of complete cavity closure.

These remarks are meant to be preliminary to what I expect to say this afternoon in the hope of combating what I believe is present overenthusiasm about pulmonary resection for tuberculosis. If thoracoplasty works as well as many of us think it does, I believe we cannot afford to substitute a high operative risk and a high percentage of spread for the remarkably good results that thoracoplasty gives in a very high percentage of cases.

DR. E. J. O'BRIEN, Detroit, Mich.—I expected to say exactly what Dr. Alexander has said, and I think some of it is worth repeating. Technically, this is a beautiful piece of work, but I do not know just how much it means, nor how much benefit either the patient or the operators obtain by needling a lot of cavities preoperatively. We all admit, and know, and the essayists themselves stated that a cavity may be blocked today and not be blocked tomorrow. It may change either by itself or during the course of collapse therapy measures.

We have all seen a number of patients with apparently blocked cavities getting larger under artificial pneumothorax, which close when the pneumothorax is abandoned. This is especially true if a phrenic is added. The change in position apparently relieves bronchial angulation. We have also seen definite tension cavities at the apex which close readily after thoracoplasty. In fact, free drainage and normal pressures may occur after the first stage thoracoplasty. I object very much to the hazardous needling of a cavity to see what the pressure is, therefore, until the collapse measures, which we have been using successfully so many years, have been tried. In the abstract of this paper the statement is made that, "A large percentage of pulmonary tuberculous cavities are 'tension cavities' and rarely close with thoracoplasty." Dr. Alexander referred to this statement, but I want to take exception to it also.

We have well over 1,000 thoracoplasty patients, and our percentage of cavity closures will run from 80 to 90 per cent. Almost any report on a group of thoracoplasties will be the same. When the number of cavities closed with artificial pneumothorax and with phrenic nerve interruption is added, there are not many left. I am afraid we are going to become tension-cavity conscious and neglect those procedures that have been effectual for so many years, for other measures more hazardous and less successful. I should stress this point as far as lobectomy is concerned. I agree with Dr. Alexander that we must remember that a small area of necrosis in a lung may be stretched by the intrapleural negative pressures to give the appearance of a large balloon cavity and a false impression of the actual amount of disease present.

It seems wrong to me to take out a large amount of good lung tissue because a small area of necrosis exists in it. This is frequently done in apparently blocked cavities.

As far as Dr. Head's paper is concerned, no one can take issue with any procedure in individuals with far-advanced, hopeless tuberculosis if it gives relief and prolongs life. I do not like the Monaldi operation but rather prefer cavernostomy in those patients in whom collapse therapy is impossible to accomplish. It often gives relief, and in some patients will cause cavity closure, but let us not go hog wild on tension cavities. There are not very many remaining after collapse methods are employed.

DR. LEO ELOESSER, San Francisco, Calif.—This difference of opinion merely shows us what I think we have been conscious of for many, many years, and that is that we do not know what we are doing when we are operating for pulmonary tuberculosis. I am merely going to discuss very briefly the pathogenesis of the tuberculous cavity and leave the question of treatment aside.

In the first place, I would like to protest at calling the cavities under discussion tension cavities. Dr. Vineberg and others have protested at my having called them blocked cavities. I think that protest is in order. If we will call them insufflation cavities, I think everyone will be satisfied. They are not tension cavities, to my idea, because "tension" connotes stretching. That is a literal translation of tension, and they are just the opposite of tension.

These insufflation cavities have two elements. They have in the first place the element of insufflation of the cavity itself. That insufflation element is not present all of the time. There is nothing, or very little, that works all of the time or 100 per cent of the time, as a very celebrated native of Illinois said a good many years ago. It is not necessary for a cavity to be under *constant* positive pressure in order to have it insufflated. All that is necessary is that it be under positive pressure more of the time than it is under negative pressure; this condition obtains in a great many tuberculous cavities, especially the round ones. What adds very greatly to the complication of the physics of these cavities is the extrinsic factor which Dr. Shipman has emphasized. These cavities are under influences not only from the inside, but from the outside, from influences that have to do with the relation of the caliber of the communicating bronchus of the cavity itself and of the bronchi that subserve the surrounding pulmonary parenchyma. And that brings us to a problem of physics that is so extremely complicated that I am afraid that I am not able even to state the questions that arise when one considers this complicated problem of aerodynamics.

It has been somewhat of a satisfaction to see how, since 1937, this topic has been elaborated. Some of the oldest inhabitants here, like Dr. Butler and others, will remember the development it has undergone, and that in 1937 so experienced a phthisiologist as our late president, Dr. Lord, stated that he had not seen enough patients with bronchial stenosis to be able to form an opinion concerning them. If, therefore, we will consider the insufflation cavity as one that is insufflated by pressures in which the positive exceeds the negative for a majority of the time, and if we will take account of the extrinsic factors, I think gradually, step by step, we shall arrive at some conclusion as to the pathogenesis of these cavities.

DR. VICTOR STRONG RANDOLPH, Phoenix, Ariz.—With Dr. O'Brien's permission I would like to mention a form of cavity drainage which I think should at least be brought to the attention of the Society. It was proposed in Toronto by Rogers and Shipman for use in post-thoracoplasty cavities, and I have used it in bilateral tuberculosis for patients in whom collapse measures cannot be used because they would so reduce vital capacity that life would not be possible to the patient, or at least not useful.

(Slide.) This was a man who was very sick. He had a large cavity in the top of the right lung and a good deal of disease in the balance of the lung. He had a small cavity in the top of the left lung and laryngeal tuberculosis; he wanted to get well. He was in bed for several months and not allowed to talk. A few months later the cavity in the right lung was a little larger and in the left lung, smaller; therefore, extrapleural pneumothorax was produced on the right side, and at a later time the cavity on the left side became very large, as can be seen, and therefore on this left side an Eloesser flap operation was performed. The next film shows that the cavity on the left side is gone, and this man, six years after his original illness, is well enough to work in a clerical capacity. This case illustrates the type of drainage that I wanted to show.

DR. LOUIS R. DAVIDSON, New York, N. Y.—I am not desirous of challenging the statement of anyone here. I do want to agree, and I do so heartily, with both Dr. Alexander and Dr. Eloesser. A few days ago, in this city, at a meeting of the American Surgical Association, Dr. McClure of the Ford Hospital of Detroit read a paper entitled, "Tannic Acid Treatment of Burns." This was an obsequy. He hoped that no one would again use tannic acid in the treatment of burns.

Regarding the Monaldi operation, as an individual procedure, I sincerely trust that we have read the obsequies. I think I am in a safe position to make this statement. Dr. Butler will tell us whether this society met in 1940 in Cleveland. At the meeting there, and all this is a matter of record, I deprecated the use of the Monaldi procedure as an individual method. The next year, in San Antonio, that statement was again made, and at this moment in no way do I feel that I want to change my opinion. I could give you my reasons for this conclusion, but I believe they are well known to you.

I had the privilege of knowing the late Dr. Pol Coryllos; I was perhaps affected by his dynamic personality and his fierce ambition. I am happy to find that today, many years after his death, pathogenesis, dynamics, and the closure of tension cavities are being discussed in almost his very words. He is now, perhaps, receiving commendations which he so well merited in this direction. To be sure, he and I disagreed very heartily, in some instances, on the question of the cavity in pulmonary tuberculosis. I agreed with him that there is a definite intracavitary influence at the bronchocavitary junction, but as Dr. Eloesser and Dr. Shipman have also stated, we insist that there are extracavitary influences. The Monaldi procedure in no way permanently influences these extracavitary factors.

I should like to show a few lantern slides. I devised this cannulating pulmonary cavity instrument some years ago in order to take frequent pressure readings while the cannula was in the lung and while we, with the aid of specially devised telescopes, could inspect the inside of these tuberculous pulmonary cavities. We know that the wall of the cavity is composed of three layers, the inner layer which is cheesy material, the middle layer or zone which is vascular in nature, and the outer or fibrous layer. This last zone is never noted upon

inspection, except by the pathologist at the autopsy table. Through these telescopes we took colored pictures of the inside of the tuberculous cavity in the living patients. In only two of the thirty-seven cases studied could we definitely visualize the bronchus.

Here is a recess which is noted in all cavities. (Slide.)

I might say that almost every cavity which is cannulated by the Monaldi technique will close, but when the catheter or cannula is permanently removed, sooner or later, the cavity reopens.

Here is a patient with a catheter inserted into the cavity. You will note the fluid level. When this patient forcibly expired and the catheter was pinched, the cavity was obliterated. That is what occurs when good, constant negative pressure is applied to the cavity through the catheter, but when the catheter is removed, the cavity reopens, due particularly to extracavitary influence. This procedure may have a place, very occasionally, when it is combined with a thoracoplasty. With this in mind, an anterior thoracoplasty was first performed. The thought occurred to us that the brachial plexus and subclavian vessels might have dropped after the first rib had been removed, thereby leaving these structures without support and permitting them to fall over the space we would use for the cannulation. In order to determine this development, diodrast was injected into the cubital vein on the same side and the patient immediately x-rayed. It was found that these structures had not changed their original positions.

Because of this fact, the Monaldi was then performed, followed by an orthodox posterior thoracoplasty.

In closing, I may say the Monaldi procedure will not be used too often. While I do not call for obsequies, I feel that its reincarnation, in any form, should be very infrequent.

MAJOR WILLIAM W. WOODRUFF.—I wish to discuss Dr. Head's paper, as our experience, based on thirty-five cases, is similar to his.

We too have used Monaldi drainage almost entirely as a salvage procedure, reserving it for those cases in which other forms of therapy did not seem wise or possible. I believe it also has a place in the treatment of the insufflation cavities described by Dr. Eloesser, where in most instances it will be used in conjunction with thoracoplasty. When it is so used the question arises, should the Monaldi or the thoracoplasty be done first. There are disadvantages to each method. Our present procedure is giving reasonably good results and is as follows. Almost every cavity is packed because we have had the experience of finding a free pleural space at the time of introducing the trocar when none had been found by the aspirating needle. At the time of packing we have performed an anterior thoracoplasty, resecting portions of the first, second, and sometimes the third ribs. Then ten days to two weeks later the catheter is introduced. This method of procedure has the advantage that it is possible to keep the catheter in place while the posterior thoracoplasty is done and still take the entire remaining length of ribs without running a great chance of breaking through into the infected sinus tract.

Our indications and results in the thirty patients in whom the catheter was successfully introduced are as follows:

Seventeen had extenesive bilateral disease. Of these seventeen, ten had either a cavity, unexpandable pneumothorax, empyema, or thoracoplasty on the opposite side.

Nine were chosen because of the acuteness of their disease.

The indication in the remaining four were age in two (58 and 60 years), refusal of thoracoplasty in one, and previous unsuccessful thoracoplasty in one.

I believe you will agree that any good results in this group can well be listed as salvage.

Now a word about our results in the group of thirty. In only four cases, or 13 per cent, have the catheters been withdrawn and the cavities remained closed without any other procedure being used.

Six of the patients, or 20 per cent, are dead. Four of the deaths were in the group of nine with acute tuberculosis. This is a high mortality rate, but an equal number (four) in

the same group are much improved. At the time of interference it seemed as though very little could be done for any of them.

In eight cases, or 27 per cent, it became possible later to supplement the Monaldi by other procedures. Six patients have had thoracoplasty, one has had lobectomy, and one has had a cavernostomy.

Twelve, or 40 per cent, still have the catheter in situ. Some of these may eventually die, but in some the condition may eventually become arrested.

Based on this experience, we agree with Dr. Head that Monaldi drainage is a valuable procedure and enables us to salvage some patients for whom we have little else to offer.

Dr. Vineberg has been able to have suction throughout his hospital. We have not been so fortunate; our patients have been scattered throughout the village, making necessary to have individual suction for them. We have developed a simple, portable cabinet unit. Suction is obtained by using a Stedman suprapubic suction pump. The pressure is controlled by using a mercury safety valve, and an anaeroid pressure gauge from a Zavod pneumothorax machine has been connected in the circuit so that the pressure fluctuation may be constantly observed. It is simple, reliable, and safe. The motor runs continuously and we are able to maintain a constant predetermining negative pressure.

DR. OTTO C. BRANTIGAN, Baltimore, Md.—I wish to emphasize that tuberculous pulmonary disease is a dynamic process. The mechanism which produces a tension cavity is not always constant or permanent. The intracavitary pressure will vary from time to time, fluctuating between positive and negative pressures. It would be ideal if collapse therapy could be applied during the phase of negative pressure within the cavity.

DR. FRASER B. GURD, Montreal, Canada.—Just a word or two with reference to Dr. Alexander's discussion regarding the contribution of Dr. Vineberg and Dr. Kunstler. He referred to the fact that in the article published by them in *Surgery, Gynecology, and Obstetrics* in March of this year, the total number of cavities obliterated in the group subjected to transthoracic intracavity drainage is small. I wish to point out that this group of cases refers only to thoracoplasties that have been done upon chests in which the tuberculous cavities have been one inch, or more, in diameter and that the more simple lesions which we prefer to treat are not included in this group. In other words, those patients who are not in the salvage group are not included in these figures. I am not personally able to state offhand the number of straightforward cases in which operation has been done, but in those patients who had smaller cavities and who were not needled, the number of closures was very high and would have raised the total number of successful cases substantially.

Dr. Vineberg and Dr. Kunstler are not, I believe, suggesting that the problem of obliteration of large cavities, whether these be positive pressure or not, has been solved. In this attitude every one of us in this room would be in agreement; Dr. Vineberg and Dr. Kunstler have, however, brought forward for consideration a method of investigation which seems to be safe and, in the main, to be very satisfactory. I know that I personally, watching the work of these younger men, have been fascinated by the information which they have obtained and pleased by the very substantial improvement of the results in our clinic since routine determination of pressure in all larger cavities has been carried out.

DR. ARTHUR VINEBERG, Montreal, Canada.—The first thing I would like to say is that we have never, even in our writings, suggested that intracavity suction drainage was a means alone of closing a cavity of the tension or positive pressure type. You speak of intracavity or extracavity factors. We, too, believe in the extracavity factors, and that is why we believe in doing an anterior stage thoracoplasty.

With regard to Dr. Alexander's remarks—it is quite true according to our figures that we did have 42 per cent of cavities which were needled and proved to be tension cavities. Ordinarily across the country, about 30 per cent of cavities failed to close after thoracoplasty and became residual cavities, that is if only cavities are considered. In reporting 1,500 patients treated by thoracoplasty with 83 per cent closure of the cavities, I would like to go

over those cases and see how many of them were simply minimal disease or small, tiny interloculated cavities. We are talking about cavities all of which are larger than 2.5 cm. This is the first point.

The next point is that I quite agree with Dr. Alexander that some of these patients may have tension cavities at the apex when we needle them. But in a certain percentage of these cavities following thoracoplasty, the bronchus becomes twisted and closed off, which would help to explain the discrepancy between the figures of 42 per cent tension cavities and 17 per cent failure of cavities to close by thoracoplasty.

Dr. O'Brien objects to needling of cavities. We have had absolutely no complication from needling in our series. We think that it is a crime to make a respiratory cripple of a man because he has one cavity. It is not the right thing to do a seven-rib thoracoplasty on a man if a five-rib operation following intracavitary suction drainage will suffice. A good healthy lung is being obliterated instead of the diseased area. We are attempting to knock out only the diseased areas and not sacrifice healthy lung in order to do so.

Dr. Davidson showed us where he had examined various cavities from bronchi and found only two bronchi in the endoscopic examination. May I point out to Dr. Davidson that the average pathologist who examines the lung does not find the tension or at least the valvular mechanism because he doesn't look for it, and we, ourselves, have gone back over cavities which were declared to be without any definite valvular mechanism in their draining bronchi, and we have found in every case a bronchus leading to the cavity with a trap-door mechanism or a bulbous type of swelling in the bronchial wall. You have to look for it, and you have to look for it at the autopsy table. It will not be seen necessarily through the endoscope.

In closing, with regard to Dr. Head and Dr. Woodruff who suggest that this method should be reserved for salvage cases, it seems to me that their cases are no criteria whatsoever of the efficiency of this method. The previously mentioned workers have attempted to salvage people. There is no point in trying to salvage patients with a vital capacity of 1,000 c.c. or less, but if such an attempt is made, from the results of Dr. Head's cases and from what Dr. Woodruff reported, I would say that it speaks very well for intracavity drainage per se. Why reserve this method for closing cavities in salvage cases? Why not try to close large cavities with the minimum amount of damage to healthy lung?

DR. JEROME R. HEAD, Chicago, Ill.—I wish to express my appreciation of the very excellent work that Dr. Riggins and Dr. Vineberg have done. I believe that it is important and will lead to improved practice. I also would express my agreement with what Dr. Alexander and Dr. O'Brien said. I believe that the truth is neither here nor there. I am sorry that Dr. Davidson did not say that Monaldi suction should be abandoned completely. It would have served as a text for a lecture. Throughout the whole history of collapse therapy and tuberculosis we have had many new techniques introduced. Eventually someone says that he has given up this or that operation as being of no value. I have found that everything that has been added—paraffin pack, extrapleural pneumothorax, Monaldi suction—each has a place; there are patients who cannot be treated in any other way. The one who practices the best will use them all as they are indicated.

PRINCIPLES OF IMPROVING INADEQUATE TRACHEOBRONCHIAL DRAINAGE FOLLOWING TRAUMA TO THE CHEST

Further Problems in the Treatment of Wet Lung

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INTRODUCTION

In A previous paper[9] the development of wet lung in patients with thoracic trauma was presented and the early therapeutic problems discussed. In most instances the application of the measures suggested is successful in "drying up" the tracheobronchial tree so that the patient is well on his way to recovery. It is true, too, that in spite of the most strenuous early treatment a number of severely wounded patients with wet lung will die. On the other hand, there does exist a definite group of patients with thoracic wounds who survive in spite of the persistence of some fluid material in the bronchial tree. There may be a recurrence of the formation of these obstructing fluid substances in the respiratory tract. In this group of patients pulmonary atelectasis and pneumonia are prone to occur.

CAUSATIVE FACTORS

Pulmonary atelectasis develops following trauma to the chest in a manner that parallels the pathogenesis of postoperative atelectasis seen in civilian life. In either situation when atelectasis is unrelieved, pneumonitis results. First, fluid substances are present in the pulmonary tree in abnormal amounts. Second, there are forces which prevent adequate expulsion of this fluid material, so that bronchial obstruction develops. As a result of extensive civilian experience, bronchial obstruction has been recognized as the important etiologic factor in the development of postoperative atelectasis and pneumonia. Therefore, it is not necessary to review this evidence at this time. However, it should be emphasized that bronchial obstruction is present in these patients, for if it is dealt with strenuously, the morbidity and mortality will be greatly reduced.

There are various types of fluid substances which may be found in abnormal amounts in the tracheobronchial tree of patients with thoracic wounds. Blood, tenacious mucus, and purulent secretions, or a combination of these may be present. The blood, obviously, comes from an area of intrapulmonary hemorrhage. De Takats[1] showed experimentally that thoracic wall trauma resulted in bronchial spasm and increased bronchosecretory activity. This is the probable explanation for the occurrence of tenacious intrabronchial mucoid secretions which are encountered with such surprising frequency following trauma. Because of the prevalence of bronchitis in soldiers under combat conditions, particularly during the winter months, purulent bronchial exudate often antedates injury. Purulent sputum may also signify a developing infection. Evidence that infection has developed may be gained by observing that the sputum which

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was at first only bloody or mucoid has become purulent. Fluid in the pleural cavity may give *bronchial* symptoms primarily. In the presence of hemothorax or empyema a constant productive cough may well be due to the continued seepage of intrapleural blood or pus into the bronchial system through a bronchopleural fistula (Case 1).

Necrotic mucosa, debris, and membranous exudate from burns of the lower airway and from the local action of certain poison gases may cause widespread tracheobronchial obstruction within a few days after injury. Following burns, autopsy findings showed extensive bronchial obstruction.[2] Bronchial plugs and inspissated casts have been shown to form due to the local action of mustard and related gases in the lower airway. Although we have had no such cases in our series, it would seem logical that bronchoscopic aspiration would be beneficial.

In thoracic injuries there are present many factors which disturb effective coughing and the successful expectoration of the various types of sputum previously discussed. Thoracic wall pain is one of the most important factors. Respiration is shallow and the patient will not cough voluntarily because of the severe pain experienced. Diaphragmatic injury, diaphragmatic paresis (? reflex), abdominal ileus, and fluid in the pleural cavity by their "splinting" effect may result in ineffectual coughing. In multiple rib fractures the associated "flail" chest and paradoxically moving thoracic wall result in a most inefficient type of cough. Oversedation with morphine, not uncommon in battle casualties, is followed by infrequent shallow respiration and drowsiness, which favors the retention of sputum in the bronchial tree.

SIGNS AND SYMPTOMS

In the presence of excess fluid in the bronchial tree there are certain cardinal signs and symptoms that presage the advent of more serious trouble. One of the most frequent findings is an almost constant, inefficient "wet" cough. Such a cough may be described as having a rattling or gurgling element which persists in spite of the fact that sputum is being raised with each effort. The wheeze or rattle continues because sputum is collecting in the bronchi as fast or faster than it is being expelled. The cough may be continuous, hacking, harassing, or even paroxysmal in character and without much expulsive force behind it. Occasionally, the cough may be nonproductive in spite of the most strenuous efforts. When small amounts of sputum are being raised with constant repeated coughing attacks, it often leads to the mistaken idea that the drainage is good, a fallacious assumption. On the contrary, this type of cough is evidence that only the most superficial secretions are being raised, rather like the splashing out of small amounts of fluid from the top of a full cup that is continually being replenished. The very fact that the cough is *continual*, even though slightly productive, is pathognomonic of inadequate bronchial drainage.

On physical examination there are two outstanding features: dyspnea and the presence of bronchial râles. If there has been extensive pulmonary trauma, dyspnea is usually a combination of various factors, but when the pulmonary damage has been minimal, dyspnea usually means a diffuse, partial bronchial

obstruction with resultant anoxia. Bronchial râles have been present in all patients. In a poorly emptied tracheobronchial tree, bronchial râles persist in spite of cough and expectoration. When they are wheezing and high pitched, they have a dry quality. They may be loud enough to be audible at the bedside and in such instances the patient appears to be having a typical asthmatic attack. In spite of the dry quality of these râles, they have disappeared after the patient has commenced raising tenacious sputum. In other patients, bubbling sonorous râles occasionally predominate with an accompanying classical tracheal rattle. When râles of this sort are present the sputum raised is frequently of a more liquid character.

Distant bronchial breathing may be heard over small areas, usually signifying patchy lobular atelectasis, but of course it may represent a pneumonic process. The diagnosis is certain if the intensity of the bronchial breathing or its location changes with forced cough.

Fever and toxicity are constantly present. While at this stage early bronchial infection may be a minor factor, the fallacy of treating all of these patients for a bronchopneumonia is obvious. The history, the constant wet cough, and widespread bronchial râles are signposts that will not be ignored in the proper evaluation of the patient's condition.

ROENTGENOGRAPHIC FINDINGS

The frequent coexistence of an extensive thoracic wall, pleural or intrapulmonary lesion makes the shadows possibly due to partial or complete bronchial obstruction very difficult to interpret. On the other hand, a considerable degree of obstruction may be present without roentgenographic evidence. When any question arises as to the diagnosis, more weight should be given to the clinical picture. Whenever possible, roentgenograms in at least two planes should be taken. In certain specific instances, collateral information may be gained by thoracentesis when it is considered in the light of suggestive roentgenographic findings. The shadows caused by atelectasis must be differentiated from those produced by pulmonary contusion or hematoma. With intrapulmonary bleeding the shadows are quite likely to be round or oval and in the case of a hematoma there may be small, loculated airpockets present. Intrapulmonary bleeding will not result per se in cardiac or mediastinal shift.

TREATMENT

With any degree of bronchial obstruction, treatment is aimed at improving the impaired tracheobronchial drainage as rapidly as possible. Cardiorespiratory imbalance from mediastinal shift, shock, ileus, etc., is corrected simultaneously. Thoracentesis is an extremely valuable preliminary step in the treatment of bronchial obstruction, and must always be performed at once whenever there is any evidence of pleural fluid. Gastric and intestinal decompression should be commenced as indicated. The development of deep full respirations and an efficient cough is mandatory.

Pain from thoracic wall contusions or from fractured ribs has been a frequent reason for inability to breathe deeply and to cough and raise sputum. In

treating a large number of thoracic casualties one of the most significant observations has been complete confirmation of the value of intercostal and paravertebral nerve block in the control of thoracic wall pain. This has been reported previously by Rovenstine and Byrd,[7] although these authors did not elaborate on the relationship of thoracic wall pain to wet lung and defective bronchial drainage. The indications, technique, and results of this simple and valuable procedure have been given.[10] Nerve block has completely relieved pain and discomfort for a period of time averaging not less than twenty-four hours. But it may be that the relaxation of voluntary muscle in the thoracic wall is important in the relief of pain well beyond the time of action of the anesthetic agent. With the exception of severe flail chest, nerve block has precluded all necessity for strapping the thorax, in itself a procedure which decreases respiratory exchange and favors the retention of bronchial secretions. Nerve block is, then, indicated at once on any patient with faulty bronchopulmonary elimination who is complaining of thoracic pain. It should be used prophylactically in all patients with severely painful chests, where respiration and cough are limited. The cough almost immediately becomes efficient and productive; dramatic improvement usually follows within one hour. Nerve block may be the only procedure necessary to aid in clearing the obstructed bronchi (Case 3).

Carbon dioxide inhalations combined with the frequent changes of the patient's position are likewise of considerable value. The gravity effect of positional change on bronchial secretions is too well known to require further description. We much prefer 100 per cent CO_2 when it is available, administering it for two or three deep breaths by means of a catheter and small funnel. Forced coughing, during and following the brief stage of hyperpnea, is of the utmost importance, a detail not often sufficiently stressed. Without forced cough, the administration of CO_2 loses much of its effectiveness. When no CO_2 is available, rebreathing into a paper bag may be substituted. The value of carbon dioxide is twofold; the gas itself is a true expectorant[3] and, mechanically, hyperventilation loosens and propels viscid secretions along the tracheobronchial lumen.

CATHETER SUCTION

Depending on the condition of the patient, on his roentgenographic findings, and on our clinical judgment as to the progress toward clearing, the measures just enumerated are continued for from six to twenty-four hours. If the patient does not progressively improve or if there is evidence at any time of further blockage, mechanical aspiration of the tracheobronchial lumen is resorted to without delay. This is performed by either of two methods: tracheobronchial catheter aspiration[4] or bronchoscopy. Each method has its advantages and there are but few clear-cut indications for the use of one rather than the other method. Bronchoscopy may be followed by repeated catheter aspirations. In both methods the basic aims are the removal of excess fluid in the lower airway, the loosening of deeper secretions by local manipulation of the tracheobronchial tree, and the development of a more efficient cough.

The equipment for catheter suction is almost always available and the technique is simple. The fact that with practice no anesthesia is necessary makes this method one that can most easily be used in an emergency. Rarely, a small amount of topical anesthesia may be required if a hyperactive gag reflex is present. The technique should be mastered by surgeon and internist alike; many lives will be saved thereby. A source of suction delivering from fifteen to twenty pounds negative pressure (ordinary "nose and throat" suction machine), connecting tubing, and a No. 16-18 new urethral catheter are all the materials necessary. A portable hand-driven suction machine has been designed by one of us (L.A.B.)[5] for use when no source of electricity is available. The Robinson type of urethral catheter with two openings is preferred. The patient is placed in semi-Fowler's position, the neck is flexed, and the tongue pulled rather sharply forward to anchor the larynx. If the patient is unconscious, the neck can be flexed by raising the head on a pillow or folded blanket. Without suction, the catheter is passed through the more widely patent nostril and advanced until the larynx is reached. The catheter is then withdrawn for about 1 cm. and the patient asked to take a rapid, deep breath. During inspiration, the catheter is quickly advanced into the trachea. Success in the manuever will be signified by coughing, passage of air through the tube, and huskiness of the voice. Occasionally it will be better to advance the catheter during the expiratory phase of a cough. After the catheter has been advanced for several centimeters into the trachea, suction is commenced. The patient is urged to cough and usually responds violently due to contact with the tracheal mucosa. The catheter is slowly advanced and retraced to provoke coughing and prevent excessive suction on the mucosa in any one spot. Suction is stopped after about five seconds and the patient allowed to take two or three deep breaths so that not all the air will be drawn out of the bronchial tree. Aspiration is then commenced again and after the trachea is dry, attention is turned to the main bronchi. The patient's head is turned slightly to the left in order that the right stem bronchus may be entered. It is somewhat more difficult to enter the left stem bronchus; the patient's head must be turned far to the right and the chin somewhat elevated. The catheter should be advanced as far as possible into each stem bronchus. Frequently, the patient is rolled on his side with the more involved lung uppermost, so that dependency will aid in draining the smaller bronchi. The continuous periods of suction should not be longer than five seconds and the patient should not be allowed to become exhausted or cyanotic. Following catheter suction the patient coughs with greater efficiency because of increased ventilation. He should be watched closely, however, for further accumulation of secretions. Since there is no trauma to the larynx or bronchi, catheter aspiration may be repeated at two- to three-hour intervals.

Certain specific uses of catheter aspiration should be listed. Acute emergencies on the ward should undoubtedly be treated by this means. When an active traumatic thoracic service is in operation it is wise to have a tracheobronchial catheter aspiration set prepared on the ward at all times. In the

unconscious patient where it is probable that frequent aspirations will be necessary, catheter suction should probably be performed from the beginning. Occasionally there is the desperately ill but conscious patient in whom tracheobronchial cleansing is urgently needed, but even the manipulation necessary for a bronchoscopy may prove hazardous; catheter aspiration may well prove a lifesaving procedure. Postoperatively, catheter aspiration is of great value if there is any question of retained secretions. Rarely, it may not be possible to pass the catheter because of an unusually flaccid epiglottis. Technically, there is no other condition which should prevent the passage of a catheter.

BRONCHOSCOPY

Bronchoscopy is a highly technical procedure to be performed by someone especially trained in this technique. General anesthesia is contraindicated in the presence of excess bronchial secretions so that topical anesthesia must be employed. Bronchoscopic aspiration is a more efficient procedure than catheter suction. Since it is done under direct vision, both stem bronchi will be aspirated in a more efficient manner than catheter suction. With the use of curved suction tips, more adequate drainage of the smaller bronchi can be accomplished. Further increase in the airway is provided by mucosal shrinkage with pontocaine (or cocaine) and epinephrine, which procedure is not possible with catheter suction.

Under certain conditions bronchoscopy is carried out at once without waiting for a trial of more conservative measures. Bronchoscopy is the treatment of choice when the obstruction is due to particulate material, blood clots, and thick mucous plugs.[8] In bilateral atelectasis the patient has a sufficient reduction in his vital capacity so that a curative procedure is indicated without delay (Case 2). In unilateral massive collapse, immediate bronchoscopic aspiration is preferable to a more conservative regimen. With an already reduced vital capacity, the sudden loosening of large amounts of secretion from the obstructed bronchus can well flood the normal bronchus with grave results. In partial tracheal obstruction an immediate bronchoscopy must be considered. Here again, there is great danger of aspirating excess secretion into the more normal bronchus. When there is sudden recurrence of obstruction after partial or complete clearing of the bronchial lumen, early bronchoscopy is demanded. At times a patient is admitted to the ward completely exhausted, without the strength to cough and with the tracheobronchial lumen brimming with secretions. Immediate aspiration is mandatory. Here is a seeming paradox, that the sicker the patient, the more necessary is an immediate bronchoscopy (Case 1). Failure to follow this principle will result in disaster. Stomach secretions have an early deleterious effect on the tracheobronchial mucosa.[6] When an unconscious patient aspirates vomitus it is an imperative indication for mechanical cleansing of the tracheobronchial tree before the patient leaves the operating room. Bronchoscopy is much the preferable procedure. Catheter aspiration must, however, be substituted if the patient has only partially reacted from the anesthesia and his neck and jaw muscles have become tight. Justification for stressing this point lies in the fact that much

of the forward surgery is of an emergency nature. Postoperative vomiting will, therefore, be a more frequent complication than in civilian surgery.

CASE REPORTS

Many patients showed a significant degree of intrabronchial obstruction following thoracic trauma. Three case histories have been chosen from this group to illustrate many of the problems of diagnosis and treatment. Patients were received at the thoracic surgery center from four to five days after injury during the Sicilian invasion and the early part of the Italian Campaign.

CASE 1.—A 23-year-old soldier received multiple machine gun bullet wounds while fighting in Sicily on July 15, 1943. The most serious injury was a right-sided thoracoabdominal wound, the missile entering the thorax anteriorly just below the clavicle and coming out through the upper abdominal wall. An emergency laparotomy was performed at a field hospital. A laceration in the right dome of the liver was found and packed. At the conclusion of the operation the patient was in critical condition from right-sided tension hemopneumothorax and an obstructed lower airway. Twenty-two hundred cubic centimeters of blood and air were removed from the right pleural cavity, followed at once by bronchoscopic aspiration of obstructing secretions. There was immediate dramatic improvement. The tension pneumothorax was treated by an indwelling needle attached to a waterseal bottle. This needle did not function during transportation. The patient arrived at the thoracic surgery center on July 19 in poor condition with dyspnea, cyanosis, and a weak rapid pulse. Expectoration of tenacious bloodstreaked secretion eased the cyanosis somewhat. The patient had a wet cough and raised sputum constantly. There were coarse râles over the trachea and parasternally on the right. Thoracic roentgenograms showed a right-sided patchy obscuration apparently due to a small amount of pleural fluid and, perhaps, scattered areas of atelectasis. On thoracentesis, only a few cubic centimeters of thin bloody fluid without odor were obtained. Immediate bronchoscopy was productive of much bloody tenacious secretion coming from the trachea and right stem bronchus. The patient raised several ounces of similar material during the next three hours and his condition improved. On July 21, the patient again experienced acute obstructive respiratory difficulty, and emergency tracheobronchial catheter aspiration *on the ward* was undoubtedly lifesaving. This material was bloodstreaked and had a faintly foul odor. Roentgenograms were repeated and the obscuration in the right upper part of the thorax had increased. Thoracentesis now revealed thin bloody foul pleural fluid, identical to that which had been aspirated. Thus, the existence of a bronchopleural fistula was established even though the roentgenogram disclosed neither fluid level nor mediastinal shift. Intercostal water-seal catheter drainage was immediately performed and there was complete loss of the tracheobronchial wetness. Several days later, open thoracostomy with rib resection gave adequate drainage. The fistula closed spontaneously and the empyema cavity eventually became obliterated.

On three occasions bronchial aspiration was a lifesaving procedure for this soldier. We are convinced that tracheobronchial obstruction may often be a factor in early death from thoracic injury. Many of these deaths can be prevented if the possibility of bronchial obstruction is not overlooked. Another feature worthy of comment in this patient is the dramatic fashion in which proper drainage relieved bronchopleural spillage as in any case where a fistula connects a hemothorax or an empyema with the tracheobronchial tree.

CASE 2.—A 23-year-old British soldier was wounded in action on the Salerno Beachhead on Sept. 9, 1943. He suffered a perforating gunshot wound of the right side of the chest. The patient's wound was dressed on the beach and he was evacuated to a Landing

Fig. 1.

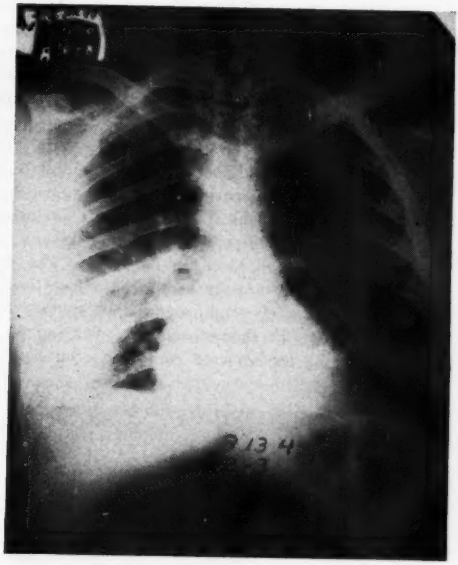

Fig. 2.

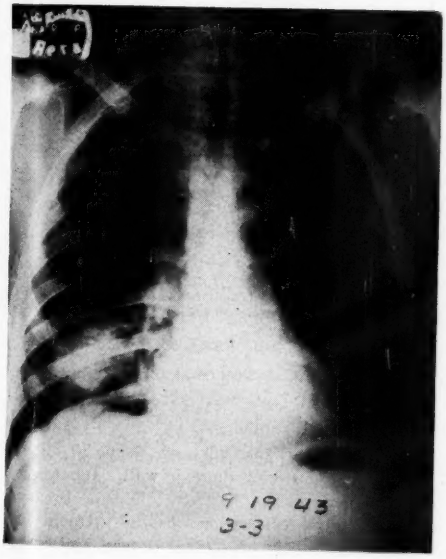

Fig. 3.

Fig. 1.—Bilateral basal atelectasis; patchy pulmonary contusion of the right side.

Fig. 2.—Sixteen hours following bronchoscopic aspiration; the atelectasis has cleared completely.

Fig. 3.—Six days following bronchoscopy; rapid clearing of the area of intrapulmonary bleeding.

Ship Tank (LST) on which he remained for three days, arriving at the thoracic surgery center on September 12. He was exhausted, dehydrated, and dyspneic and he had a wet cough without expulsive force. He was raising only small amounts of faintly bloody mucopurulent sputum. Physical examination showed no shift of the mediastinum. Coarse

rhonchi were heard over the midline. At the base, bilaterally, were areas of faint bronchial breathing associated with fine râles, which shifted slightly in location and intensity following cough. Roentgenograms (Fig. 1) showed a bilateral basal triangular shadow extending down and out from the hilum and considered typical of bilateral atelectasis. Peripherally, at the right base, the rounded area was interpreted as probable intra-alveolar bleeding (pulmonary contusion).

Bronchoscopy was performed several hours after admission because of the patient's extensive bronchial occlusion, lowered vital capacity, inability to cough efficiently, and exhaustion. Obstructing tenacious mucosanguineous secretions were aspirated from both lower lobar bronchi. After mucosal shrinkage, the patient coughed up quantities of tiny pluglike casts composed both of blood and of mucoid material. These obviously had been obstructing small bronchi bilaterally. The patient was immediately relieved of the dyspnea and the breath sounds at the base lost their bronchial quality. The cough was productive of approximately eight ounces of sputum during the next eighteen hours. At this time roentgenograms showed complete clearing of the bilateral atelectasis (Fig. 2). Progressive clearing of the area of contusion occurred in the next six days (Fig. 3).

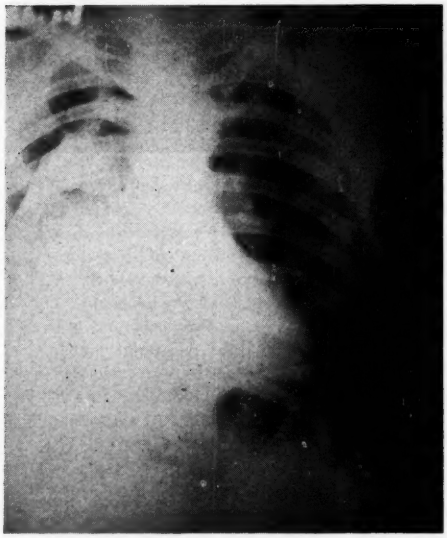

Fig. 4.

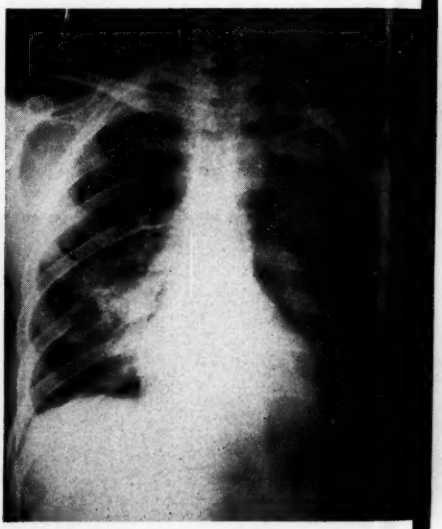

Fig. 5.

Fig. 4.—Right-sided hemopneumothorax; probable partial bronchial obstruction: the seventh and eighth ribs have been fractured posteriorly.

Fig. 5.—Extensive clearing twenty-four hours later.

CASE 3.—A 27-year-old soldier was wounded in action in Italy, on Sept. 13, 1943. He suffered a perforating "sucking" wound of the right posterior thoracic wall. Immediate treatment consisted of a massive occlusive dressing and plasma by vein. Within twenty-four hours the patient had been admitted to a semimobile evacuation hospital. Operation consisted of wound trimming, removal of rib fragments, repair of pleural defect, and tight skin closure. The patient was having constant hemoptysis at this time. Because of tension pneumothorax an indwelling needle with flutter valve was inserted in the second anterior interspace. It was removed in seventy-two hours. The patient arrived at the thoracic surgery center on the evening of September 18. He was in no respiratory distress and was only slightly dyspneic on exertion. Physical examination showed a right-sided hemo-

pneumothorax without mediastinal shift. The wounds were clean. Early next morning the patient suddenly developed great respiratory distress. He commenced to perspire, became moderately cyanotic, and developed a constant wet, painful cough, productive of small amounts of tenacious mucoid sputum. The pain was limited to the posterior thoracic wall on the right. Loud coarse rhonchi were heard over both lung fields posteriorly. Roentgenograms, taken immediately, showed a hemopneumothorax on the right with approximately 30 per cent compression of the lung (Fig. 4). In addition, there were patchy areas of pulmonary obscuration which were difficult to separate from the shadows due to the intrapleural fluid. Because of the patient's obvious distress and the bronchial obstruction, it was tentatively decided that bronchoscopy should be done. First, however, right posterior intercostal nerve block was performed, from the fifth to the tenth thoracic vertebrae inclusive, and 1,200 c.c. of bloody fluid and air were removed from the pleural cavity. Thus, pain and the splinting effect of pleural fluid were removed. Almost immediately the patient was able to breathe more deeply and to cough effectively. Within a short time after productive cough started, the patient stopped perspiring and the bronchial râles disappeared. He raised six ounces of extremely tenacious mucoid sputum during the next five hours. The efficient productive cough, aided by small amounts of CO_2 continued for three days and gradually subsided. On September 20, thoracentesis was productive of 60 c.c. of old blood and air. Shortly thereafter a roentgenogram showed remarkable clearing both of the pleural cavity and of the pulmonary parenchyma (Fig. 5). The intrathoracic abnormalities disappeared completely in a few days. On the eleventh day after injury, skin separation of the wounds occurred because of an extensive deep serum collection. The pleural closure remained tight.

DISCUSSION

The tracheobronchial tree has as its primary function that of providing an airway from the alveolus to the outside atmosphere to permit the exchange of gases necessary for the maintenance of life. However, one must remember that it has other important functions as well. There is also a definite secretory function and the capacity to rid itself of the secretions, other fluid substances, and aspirated material that may be abnormally present within its lumen. The action of the cilia and the peristalsis of the bronchial musculature effect this cleansing of the bronchial tree. These intrinsic actions of the bronchi and trachea are aided by the voluntary cough mechanism. Little is known about the effect of the chest trauma on ciliary action of the mucosa and bronchial peristalsis. One might presume, however, that these functions would not be normally active. Nevertheless, there is definite clinical evidence that the normal cough mechanism is seriously impaired when there is even moderate thoracic trauma. Thus, the interference with the emptying of stagnated fluid substances in the bronchial tree results in bronchial obstruction, which if unrelieved may be followed by pulmonary atelectasis and pneumonia. With this concept of the function of the bronchi and the derangements thus brought about by thoracic trauma, the treatment then becomes simple and logical. The measures advocated are mainly those to improve inadequate tracheobronchial drainage.

It is probable that varying degrees of pulmonary atelectasis may be present early in these patients, although the diagnosis cannot be made early in most instances. This is due to several reasons. The evidence of large amounts of fluid in the pulmonary tree, shock, severe dyspnea, and anoxia dominate the clinical picture soon after injury. Furthermore, one is forced to proceed,

vigorously treating the patient without waiting for a roentgenographic examination. In fact, therapeutic measures must be employed to make the patient fit for x-ray examination. We have suspected pulmonary atelectasis early in these patients but have not had a clinical picture nor the confirmatory roentgenographic evidence. Vigorous treatment was instituted promptly on the day of injury and the days immediately following injury, without resorting to x-ray studies. Although we have suspected atelectasis soon after injury, actually the diagnosis has been made with certainty a few days after the wounding. The question of just when pulmonary atelectasis first develops in these cases will be solved when it is possible to take serial portable chest x-rays. The progress of the patient should be plotted soon after the initial trauma and at hourly intervals if necessary. However, in the forward medical installations where such facilities are not available this question is of academic importance, if one proceeds, vigorously treating the patient according to the principles that have been outlined.

CONCLUSIONS

1. Bronchial obstruction may be present in patients with chest injuries as a result of retained fluid substances in the pulmonary tree.

2. Persistent bronchial obstruction is an important factor in the development of pulmonary atelectasis and pneumonitis following thoracic trauma.

3. The principles of improving inadequate tracheobronchial drainage are discussed.

4. Illustrative case histories are presented.

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CERVICOMEDIASTINAL LYMPHANGIOMA WITH CHYLOTHORAX

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THE occurrence of chylous effusions in the chest and abdomen has been noted sporadically in the literature over the past 300 years. Their rarity has stimulated authors to report individual experiences. Recently these reports have been of more significance because of various therapeutic measures now being instituted in attempts to save the lives of patients with chylous effusions. The case to be presented does not offer a new therapeutic problem but warrants report because of the unusual nature of the basic lesion and because of certain implications which may aid in the clarification of some previously unexplained cases.

In a consideration of chylothorax, two major groups of etiologies, the traumatic and nontraumatic, are unrelated. The cases due to trauma constitute an interesting subject in many respects, such as the numerous ways in which trauma can give rise to extravasated chyle and the problems of management which arise. They are not the subject of the paper, but, for the sake of some completeness, a few of the salient points are worthy of note.

The traumatic cases present a rather characteristic sequence of events after the precipitating injury. There is usually a quiescent period (from four to six days up to two months) following the injury before pulmonary symptoms appear. This free period is more or less suddenly interrupted by the onset of dyspnea, often a severe shocklike picture, and frequently cyanosis. Chest pain may or may not be a feature. On examination, the patient will be found to have a massive pleural effusion on one or possibly both sides, and aspiration then establishes the diagnosis with the removal of a milky fluid. When sufficient fluid is removed, dyspnea and cyanosis are relieved and the patient is quite comfortable. Then the problem of management arises because the effusion generally reaccumulates rapidly, again causing respiratory embarrassment. With slow leaks requiring only infrequent aspirations, the patient may carry on for weeks or months. But if the accumulation is rapid and persistent, dehydration and emaciation are likely to develop and death may occur in a few days to a few weeks. The most serious loss is that of proteins in the chyle, and measures to diminish this must be instituted.

The types of trauma which have been responsible for chylothorax include hyperextension injuries, penetrating wounds, fractured ribs and vertebrae, and accidental lacerations occurring in the course of surgical procedures. Other traumatic types may result from coughing spells, convulsions, blast injuries, and injury to infants at the time of delivery or shortly thereafter. The most common type is the hyperextension injury from falls, bruises, etc., in which the thoracic spine is suddenly and acutely extended. Under these conditions the right crus

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of the diaphragm is said to compress the rather fragile thoracic duct against the vertebrae, causing a laceration. The chyle then leaks into the mediastinal soft tissues (or lower, giving rise to chylous ascites) in a retropleural position. A large collection of chyle gradually accumulates here until a rupture through the parietal pleura occurs due to pressure or tension. The sudden release of all this fluid compromises lung and mediastinum and produces the clinical picture to which reference has been made. It would not occur if an intact pleura were present as might happen in penetrating wounds or fractured ribs. This fact has been established by the demonstration of "retropleural chylomas" in the mediastinal tissues at operation, at post-mortem examinations and by x-ray examination.[2, 9, 11, 12] Brown in 1937 proposed drainage of these chylomas through a paravertebral, extrapleural approach to obviate pulmonary complications.[12]

Traumatic chylothorax occurs most frequently on the right side due to the anatomy of the thoracic duct (which arises from the cisterna chyli to the right of the midline and opposite the body of the second lumbar vertebra). It enters the thorax to the right of the midline, passes beneath the right crus of the diaphragm and maintains this position in the posterior mediastinum up to the fourth thoracic vertebra, where it courses to the left side and ascends into the root of the neck for a short distance, bends and enters into the left subclavian vein at the angle between it and the internal jugular vein. This termination is not constant and may consist of several branches, all entering this vein or some going also to the left internal jugular or the right subclavian vein, or both.[1]

Therapeutic measures have been conservative in the main. These consist of aspirations of fluid as often as is necessary, depending on the patient's respiratory status; low fat, high carbohydrate and protein diets; or nothing by mouth with intravenous fluids only for a time. More recently, patients have been treated with intravenous infusion of some of the chyle in an attempt to offset the great loss of proteins, particularly in prolonged leakage. Anticoagulants have been used (citrate and heparin), and with bacteriologic controls this material has been infused even when four days old without reaction. Some untoward reactions have occurred, however, and at least one death is recorded, due presumably to anaphylaxis.[7] Plasma and blood transfusions are valuable in maintaining fluid equilibrium. Other measures have included pneumothorax after aspiration, phrenicectomy, external drainage, and injection of irritating solutions into the pleural cavity to promote adhesions.

From experimental work it is known that there is a large collateral bed for the thoracic duct circulation; hence, ligation of the leaking duct would seem to offer a hopeful possibility. However, even ligation of both ends after surgical trauma failed to prevent chylothorax in a recent patient with trauma.[7] One can assume that ligation in patients with trauma is too sudden for collateralization because at autopsies revealing generalized malignant metastases, extensive blockage of the thoracic duct and many of its branches has been found without chyle leakage.

Mortality rates average 50 per cent for the patients with conservative treatment. Surgical intervention, such as ligation and repair attempts, produces a higher rate—up to 100 per cent, according to one article.[9] Operative leakage is attended with a 10 per cent mortality, and in eight bullet wound cases a 25 per cent mortality is reported.[15]

The subject of nontraumatic chylothorax has been thoroughly presented by Yater,[1] who found about 100 cases mentioned before 1935. The most common etiology is metastatic malignant disease which gives rise to obstruction to the duct and/or its radicles by implants within the lumen or by pressure on the duct from adjacent nodes in the supraclavicular area. There are included abdominal carcinomas, lymphosarcoma, and Hodgkin's disease. Tuberculosis causing obstruction of the thoracic duct is next in importance and is most common in children in whom pressure by lymph nodes or intrinsic obstruction is the cause of leakage. Other miscellaneous causes listed by Yater included lymphangiectasis, localized thrombosis of the subclavian vein in the region of its junction with the duct, filarial obstruction, and erosion by aneurysms.

Fehr[1] proposed the theory that slow obstruction would not cause an effusion unless many of the collaterals are likewise blocked (by carcinoma for instance). The reason chylous effusion is not seen more often in terminal malignancy is that death no doubt occurs before obstruction of this extent can develop.

Prognosis in patients with nontraumatic chylothorax depends on the nature of the underlying disease and the influence of therapeutic measures, such as radiation for lymphoblastomas.

Some interesting points about chyle may be mentioned. In the past, considerable effort was made to distinguish between true chylous effusions and so-called pseudochylous fluid. The latter was supposed to be milky fluid resulting from alterations in long-standing effusions in body cavities associated with malignancies, tuberculosis, cirrhosis and nephritis. The color change was said to be due to the appearance of a lecithin-globulin combination, and by means of laboratory tests the two fluids could be distinguished.[35] The main differences were the absence of fat and a specific gravity below 1.012 in pseudochylous fluids. More recently the existence of pseudochyle has been questioned.

The finding of fat in an effusion is now considered to be evidence of its chylous nature. Brescia,[4] quoting from Gandin, states that the milky appearance is due to the presence of emulsified fat and that chyle is the only source of such emulsified fat. This would appear to hold despite an observation attributed by Gerster[23] to Henschen to the effect that chyle and lymph are very similar in a starving animal and that lymph after fatty meals (for example, lymph from an extremity) contains fat in practically the same percentage as chyle-bearing lymphatics.

The nature of chyle with its fat content makes the staining reaction with sudan III of diagnostic importance. In our case and in several cases in the literature, the chyle did not stain. This does not rule it out as chyle but does require explanation. Sudan III does not stain neutral fat globules which often constitute from 70 to 87 per cent of the total lipids of the chyle of experimental animals.[17] Thus the constituents which should react may not be in a concentra-

tion sufficient to be detected. Briefly then, chyle has a milky appearance, has a specific gravity of over 1.012, contains emulsified fat which usually can be recognized with sudan III, and is sterile, and, if the fluid is alkalinized and shaken with ether, it will clear.

Cytologic studies of the chylous fluids and of peripheral blood smears have given some interesting findings.[17] Thirteen peripheral blood studies on ten different patients reported on in the literature showed six to have a high percentage of lymphocytes and seven to have a range of from 6 to 16 per cent. The cells in chyle have consistently shown a high percentage of lymphocytes, and experimentally this was confirmed by average cell counts of 20,700 per cubic centimeter, with from 85 to 90 per cent lymphocytes. Along with these findings, peripheral blood smears after ligation of the thoracic duct revealed a drop in the number of lymphocytes and eosinophiles in spite of the occurrence of a leucocytosis. Finally, it was noted that the milkiness of chyle varied directly with its fat content.

It is stated in numerous articles that the average rate of flow of chyle in the thoracic duct is from about 130 to 195 c.c. per hour, with marked variations occurring at mealtime.[1] Chyle is a mixture of liver and intestinal lymph with variable protein and fat content, depending on the relative mixture of high protein liver lymph with the bowel drainage which contains a variable concentration of fats. The thoracic duct is equipped with a valve near the subclavian junction which opens toward the vein. It has been observed that chyle has a rhythmic variation in its flow, the result of numerous factors, including intestinal contractions, contractions of the cisterna chyli and the thoracic musculature and the movements of respiration.[4] Experimental ligation of the thoracic duct in dogs rarely causes chylothorax. Only after the superior vena cava was ligated could chylothorax be produced in 50 per cent of the animals.[1]

It has been mentioned that chyle is strongly bactericidal. Furthermore, it is frequently observed that chyle can remain at room temperature for weeks without putrefaction. It can therefore be assumed that infection of chylous effusions will occur rarely and that appropriate surgical efforts to solve the problem can be contemplated in patients with traumatic cases without fear of infection.

CASE REPORT

G. C. (No. 516387), a 32-year-old man, was admitted to the Mount Sinai Hospital for the first time Feb. 8, 1944, giving a past history of episodes of crampy abdominal pains for the past ten years associated with dietary indiscretions. The last attack was from four to five months before admission, at which time pain was almost immediately relieved by medication. Four years before, gastrointestinal films at another hospital were reported to be negative.

Three weeks before admission he developed an upper respiratory infection characterized by cough with very little sputum, general malaise and fever. He was considered to have a virus infection, possibly a virus pneumonia.

Two days before admission he noted a swelling in the left supraclavicular area without local pain or tenderness. That evening he developed generalized abdominal pain, most marked on the left side. The next day the pain was worse, now localized to the epigastrium and left upper quadrant. There were no nausea, vomiting or bowel changes and no further change in the neck swelling. There was no history of trauma. It is of interest to note a previous

episode of left supraclavicular swelling which had occurred four months before, lasting several days with questionable associated mild abdominal pain.

Physical examination revealed a well-developed, healthy man complaining of severe abdominal pain. The temperature was 100° F.; pulse, 90; and respirations, 24. A soft, compressible nontender swelling was noted in the left supraclavicular area. The left lung base was dull, with diminished to absent breath sounds and no râles. Diaphragmatic excursions were poor. The heart was normal, and the blood pressure was 120/80. The abdominal wall was spastic and extremely tender in the epigastrium and left upper quadrant. No masses or borborygmi were noted. The rest of the physical examination, including a rectal examination, was negative.

The impression on admission was that the lesion might be a penetrating peptic ulcer, or possibly acute pancreatitis. Spontaneous pneumothorax was suggested but ruled out by the examination. Gastric aspiration removed 150 c.c. of clear fluid, with free acidity of 10 per cent and total acidity of 55 per cent.

Laboratory studies showed a hemoglobin of 78 per cent, subsequently rising to 100 per cent, and a white blood count of 13,500, with 72 per cent polymorphonuclear leucocytes. The urine was negative and the stools were guaiac negative on several occasions.

Conservative treatment was instituted, and a flat plate of the abdomen taken shortly after admission showed several loops of bowel in the left upper part of the abdomen to be distended with gas. In addition, a pleural effusion was demonstrated at the left lung base. An abdominal puncture was then done in the left upper quadrant, with removal of a droplet of fluid revealing a few polynuclear cells but no bacteria in spreads. Then chest aspiration was done through the ninth interspace in the left posterior axillary line and from 10 to 15 c.c. of a white milky fluid were easily removed. It had no odor, and on smear showed a moderate number of cells, mostly lymphocytes in a fibrin network on a background of tiny globules. Sudan III failed to stain these particles. Analysis of similar fluid removed subsequently from the neck revealed a total lipid content of 1.4 per cent, demonstrating the presence of chyle.

Because of the thoracic findings, the patient was transferred to our service.* He was observed and showed rapid subsidence of the symptoms until only the cervical swelling remained. The latter was aspirated and chylous fluid was withdrawn. It again failed to stain with sudan III. Culture revealed no growth. At this point one of us (H. N.) postulated the presence of a cervicomediastinal lymphangioma extending into the posterior mediastinum, and the subsequent procedures were carried out in attempting to establish this diagnosis.

Aspiration of the left chest no longer revealed fluid. Bronchoscopy was negative. The supraclavicular swelling had subsided, but despite this the area was reaspirated and 10 c.c. of the same milky fluid were again removed. (The analysis of this fluid showed 1.4 per cent total lipids and 52 mg. per cent cholesterol.) With the needle in place, 10 c.c. of iodized oil were injected with the hope of outlining the postulated structure. Roentgenograms taken over a period of two weeks showed migration of the iodized oil upward in the root of the neck, laterally toward the left axilla, and medially into the mediastinum. This movement of the iodized oil suggested its presence in preformed tissue spaces.

The patient now was completely asymptomatic and afebrile, but a definite diagnosis appeared to be warranted from the viewpoint of possible recurrence, possible therapy and also prognosis. There was no suggestion of underlying neoplastic disease and stools were guaiac negative on several occasions. It was therefore decided to explore the left supraclavicular region. This was done Feb. 28, 1944. No chylous fluid was encountered, but deep behind the clavicle a diffuse fatty mass, slightly whiter than normal and with occasional vesicles interspersed, was encountered and partially removed for study. The subclavian vein and thoracic duct were not exposed.

The microscopic examination† revealed areas of cystic dilatation with surrounding areas of lymphoid tissue as seen in cystic lymphangioma.

*We are indebted to Dr. Ralph Colp for the privilege of further study and treatment of this patient, who was originally admitted to his service.

†By Dr. S. Otani and Dr. P. Klemperer.

At the time of discharge of the patient Mar. 5, 1944, x-ray examination of the chest showed disappearance of all fluid and no further migration of iodized oil. When seen one month later at follow-up, he was asymptomatic and ready to return to work. He was symptom free one year later.

DISCUSSION

This case is of unusual interest from several standpoints. As well as can be determined from a review of the literature, this case is the first of its kind to be reported. We have found significant facts from various individual sources which, when pieced together, lend support to an interesting explanation of this picture and suggest that several cases previously described as chylothorax and chylous ascites of unknown etiology may have been of a similar nature.

The studies made and the operative findings indicate that there were tissue spaces in the neck and mediastinum which were in continuity, and with the pathology report we are warranted in calling the lesion a cervicomediastinal lymphangioma. The finding of 1.4 per cent total lipids in the aspirated fluid in the neck and pleura establishes this material as chyle; consequently the structure must be in communication with the thoracic duct itself or radicles of this channel. Knowing these facts, we then have to explain the association of supraclavicular swelling with chylous effusion in the left thorax.

In a discussion of the lymphatic system, several pertinent points concerning lymphangiomas are made.[3] The most important types of these structures have a cavernous or a cystic character and, as a whole, they apparently are subject to infection, either in part or in entirety. They may be located throughout the body in association with lymphoid tissue, superficial or deep, but are most frequently found in the neck, the mesentery, or the pelvis, in which regions the lymphatic sacs, which serve as the primitive depot for the system as later developed in the adult, are found. Infections, when they occur, are most often due to streptococci. They tend to occur in a periodic manner, generally give rise to swelling of the tumors, and occur most frequently in the caverous type of lymphangioma.

The tumefaction is the result of an inflammatory reaction in response to infection causing a disturbance of fluid dynamics within the structure. Consequently, stasis with back pressure develops, giving rise to distention of the tumor which frequently progresses to rupture of some weak part. Then the leakage of the cyst's contents will produce changes, depending on the location of the tumor. If near the skin, they can lead to swelling, discoloration and vesicles which rupture, leaving chronic "wetting" sores (lymphorrhea) with a sievelike character to the skin. If adjacent to pleural or peritoneal cavities, rupture can cause hydrothorax or ascites. When the diet is rich in fat, the fluid can take on a chylelike character. This would depend on the connections of the tumor, and in this respect there has been described a continuity of these tumors with larger lymphatic channels.[3]

In the light of these facts, we note that our patient had at least one previous episode of supraclavicular swelling and that the last one was preceded by an upper respiratory infection of almost three weeks' duration, continuing prac-

tically to the onset of the acute illness. The abdominal pain developed after the recurrent swelling had been noted. From the finding of left pleural effusion, the abdominal picture seems adequately explained as referred. The finding by abdominal puncture of a small amount of abdominal fluid containing polynuclear cells is consonant with the picture of a bland sympathetic reaction to an inflammatory process. Therefore it is likely that the upper respiratory infection gave rise to regional lymphangitis involving the tumor and setting up the cycle of events, causing a leak of the cyst's contents from an area adjacent to the parietal pleura into the pleural cavity. Then the final point is that this lymphangioma must arise from the thoracic duct or a branch because of the chylous nature of the effusion.

Further observation of this patient in the follow-up clinic may show that this series of events may be repeated following infections around the head and neck. No local therapy was employed because of complete subsidence of symptoms. The future course may require obliteration of this tumor by such measures as sodium morrhuate injections or radiotherapy, but until necessary, if ever, intervention would be out of order.

To carry the discussion somewhat further, it is possible with the foregoing case in mind to suggest an explanation for some cases of chylothorax and chylous ascites reported in children in which an etiology could not be found. In addition to the usual classification of chylothorax as traumatic or nontraumatic in origin, there are recorded several cases of spontaneous chylothorax for which no etiologic explanation could be found. This type of spontaneous occurrence has been noted in infants up to 1 year of age, and in this age group most of the cases are said to be of this type.[4] It has been suggested that there might have been an underlying congenital defect in the thoracic duct systems of these patients because of the marked grouping of such cases in this early period of life. Brescia[4] noted that of eight newborn infants with spontaneous chylothorax, five recovered and three died. The other nontraumatic cases in children are due mainly to tuberculosis.

Jennings and Rich[31] reported on a 9-month-old boy who had a severe cold followed in two weeks by rapid and difficult respirations which continued for about four weeks before a right hydrothorax was recognized and tapped, with the removal of chyle. This child did reasonably well despite recurrent effusions necessitating seventeen thoracenteses in one year, after which time the fluid began to reaccumulate more slowly. This patient's tuberculin reaction was negative and he was slowly gaining weight at the time of the report.

Brescia's[4] case was a 13-month-old boy with the clinical picture of a pneumonia showing rapid response to sulfapyridine. Four days later the child vomited and began to have diarrhea and abdominal distention. Twelve days after the onset of infection the child began to cough and breathe "hard." X-ray examination confirmed a right pleural effusion which proved to be chyle when aspirated. The leak subsided shortly following three aspirations with the removal of a total of 900 c.c. of chylous fluid which contained 3 per cent fat and stained with sudan III. The author postulated a congenital defect of the thoracic duct with a "give" after coughing or straining.

A third case of an 8-year-old boy reported by Huber and Silver[29] is also of interest. There developed a pronounced nasal discharge, malaise and fever, followed in one week by vomiting and anorexia, and four days later by abdominal swelling and scrotal edema. He had ascites with edema of the back, thighs and scrotum. Several paracenteses removed milky fluid which contained fat on chemical analysis. Exploratory laparotomy was finally done because of rapid reaccumulation of the ascites, and the intestinal lacteals were found generally dilated throughout the gastrointestinal tract, along with enlarged, spongy glands in the mesentery. To facilitate drainage of peritoneal fluid, several large strands of silk were thrust through the parietal peritoneum into the soft tissues of both thighs and postoperatively the ascites did not recur.

In summary of these three cases, a common sequence of infection, followed in from one to two weeks by an effusion of chylous fluid in the thorax or abdomen, is seen, and at the time of report, two patients had recovered, with the third improving. No definite etiology could be found in those with chest involvement, while at laparotomy in the last patient, generalized obstruction and dilatation were noted in the abdominal lymphatics and could have represented the result of an old inflammatory process causing obstruction. Therefore we might postulate a congenital lymphangiomatous structure somewhere along the course of the thoracic duct in all three patients.

The occurrence of lymphangiomas in the neck is well known as exemplified by hygroma cysticum colli congenitum. But these are not always confined to the neck. They have been found to extend through muscle planes of the chest wall, up toward the jaw, and down behind the sternum into the anterior mediastinum. Dowd[28] reported two cases of lymphangioma extending into the mediastinum (1913), Singleton[24] added two more, Watson and McCarthy[21] mentioned their occurrence, and, most recently, Arnheim[22] reported two extensive cervicomediastinal lymphangiomas treated by surgery and injection of morrhuate. All of these cases were characterized by swelling with or without pressure symptoms.

SUMMARY

1. There is reported a unique case of cervicomediastinal lymphangioma, presumably arising from the thoracic duct or some of its radicles, which ruptured, causing a left chylothorax. The problem of diagnosis is discussed.

2. From a study of the case and a survey of the literature, a similar pathology and sequence of events in several previously reported cases of chylothorax of unknown etiology are postulated.

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A MUSCULOPLASTIC INCISION FOR POSTERIOR THORACOPLASTY

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THE usual incision for posterior paravertebral thoracoplasty is carried straight down through the skin and underlying muscles to the ribs, making a J-shaped flap which is reflected forward. This method of making the incision has the following disadvantages:

1. It cuts through the thick muscle mass formed by the trapezius and rhomboids in the vertical part of the incision (Fig. 3) and through the latissimus dorsi and part of the serratus magnus in the lower part. Such willful section of big muscles offends the surgical conscience.

2. The hemorrhage, even under local anesthesia, is often considerable, is always undesirable, and may be so large in amount as to constitute a definite added risk in a feeble patient. The posterior scapular artery, as it runs down from above on the deep aspect of the rhomboids, is variable in position and may easily be divided.

3. That part of the muscles which lies between the incision and the spine must have its nerve supply cut, and almost certainly becomes atrophied and fibrous.

4. Much of this medial strip of muscle is indifferent material to hold a suture; the rhomboid layer is certainly of poor quality after division, and from the level of the fourth spinous process upward, the trapezius is aponeurotic and its fibers run largely transversely so that sutures obtain a poor grip. This medial strip of the aponeurotic portion of the trapezius probably has a poor blood supply and may easily undergo ischemic necrosis (Fig. 4).

5. It is particularly in this highest part of the incision that the wound is likely to give way when the stitches are removed. If this happens, it will usually be seen that the muscular layer had given way earlier, leaving the skin to take the whole strain.

6. If the wound does open, the direct superimposition of the skin and the muscle suture lines means that the extrafascial space is opened.

7. Although proper attention to the movements of the shoulder and scapula should result in no appreciable disability, it is difficult to believe that the muscles have not suffered permanent damage from the long incision.

8. The incision through the muscles is repeated at the second stage with consequent infliction of further injury.

For the last year I have been using a simple musculoplastic modification of this incision which seems to avoid most of these disadvantages. It is a simple and logical step and has proved very satisfactory in practice.

DESCRIPTION OF INCISION

The usual J-shaped incision is made down to, but not through, the muscles of the back. The vertical limb should be placed from two to two and one-half

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fingerbreadths from the midline. The medial flap is now undercut as far as the midline for the whole length of the vertical limb; the undercutting is continued into the first three to four inches of that part of the incision which curves forward, typically to just beyond the anterior border of the trapezius muscle. This undercutting is carried downward and medially for some two inches to expose the lowest part of the trapezius to within two inches of its lowest origin, where it is little more than one and one-half inches wide (Fig. 1). The most anterior part of the incision need not be undercut, but its lower edge should be retracted before the latissimus dorsi is divided. The incision in the latissimus dorsi is carried back to the lowest exposed portion of the trapezius, and this muscle is then cut through transversely to the midline. A finger is next inserted

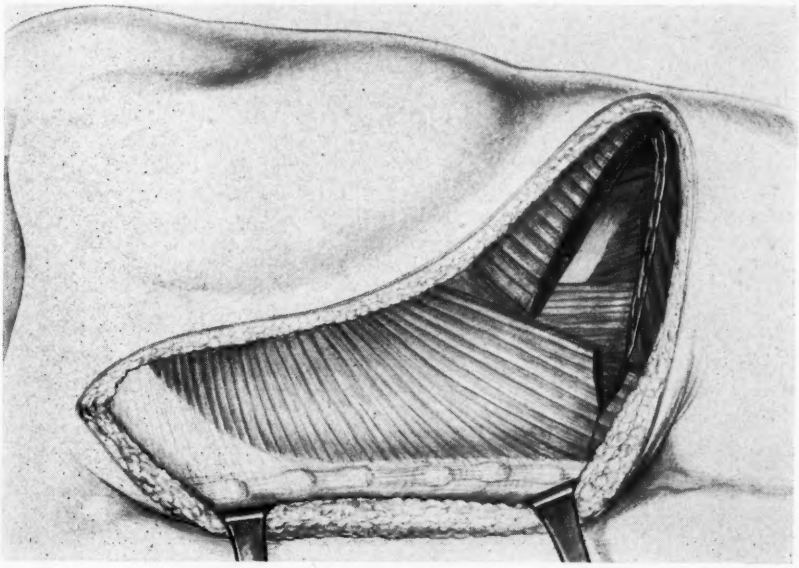

Fig. 1.—The vertical limb of the incision is shown undercut and retracted to expose the trapezius as far as its origin from the spine and spinous ligaments. Note how low the trapezius is divided.

beneath the trapezius, immediately next to the origin from the spinous processes, care being taken to separate the muscle from the aponeurosis covering the erector spinae mass, to which it may be moderately adherent. With scissors or a knife, the trapezius is cut as near as possible to the spinous processes and interspinous ligaments (Fig. 2). With the finger separating the muscle in front of it, the division is carried right up to the top of the wound. The rhomboid muscles are, of course, raised with the trapezius when they are reached, and the serratus posterior superior may also be included, or left on the ribs, if desired. In the lowest part of the incision, the muscular fibers of the trapezius come so near to the midline that there is only a very narrow strip, barely 0.5 cm. wide, of aponeurotic tissue to be divided (Fig. 3). Higher up, this aponeurotic origin

is slightly wider; the rhomboids contribute a very thin lamella to it. From the fourth spinous process upward, the aponeurosis rapidly widens (Figs. 2 and 4). As the aponeurosis is being cut, a little care will enable one to see and secure the medial divisions of the posterior branches of the intercostal vessels, thus further minimizing blood loss. The whole muscle flap is now displaced forward in the usual way (Fig. 2). As it is raised, the lateral divisions of the posterior branches of the intercostal vessels are displayed and can be secured and divided without blood loss. If the serratus posterior superior has been lifted with the flap, it can now be separated from its insertion into the ribs.

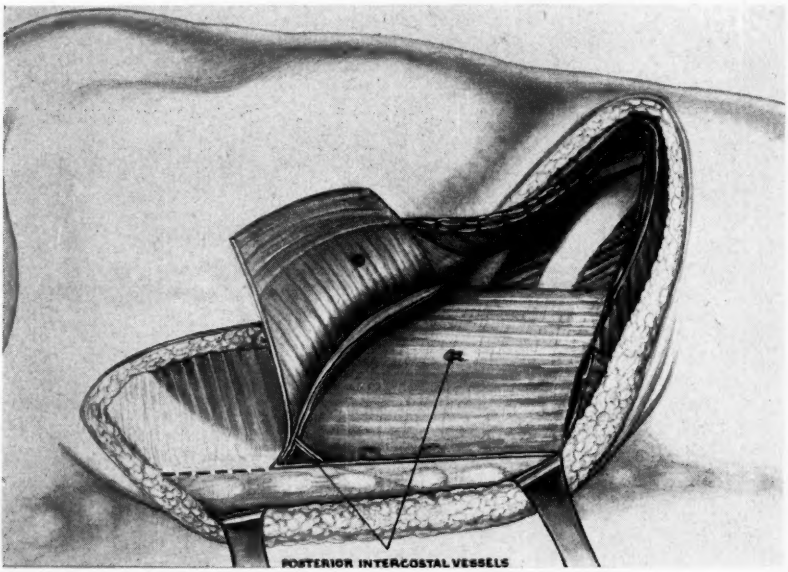

Fig. 2.—The trapezius-rhomboid layer is being freed by division of the aponeurotic origin as close as possible to the spinous processes. Note how the posterior intercostal vessels can be identified and secured before their division.

At the end of the operation, the reflected muscle layer is sewed back to its original attachment to the spinous process and the supraspinous and interspinous ligaments. This is an easy step, and the stitches find an excellent hold in the spinous ligaments. This is especially so in the highest aponeurotic portion of the flap. Only one layer of sutures is needed instead of the usual two layers, although a few interrupted reinforcing sutures may be inserted if needed.

At the second stage the maneuver is repeated; the separation is made with ease and there is very little "gumming" together of the layers, as is seen when the muscles have been cut through. The aponeurosis can be cut from the midline as easily as at the first stage and can be resutured into position just as easily and securely. The separation of the muscle flap is almost entirely bloodless at the second stage.

The advantages claimed for this musculoplastic incision are as follows:

1. A long, unsurgical division of muscle tissue is avoided, with consequent diminution of blood loss, interruption of nerve supply and subsequent impairment of function from atrophy and fibrosis.

2. The posterior scapular artery is completely out of danger when the highest part of the incision is made.

3. The exposure is definitely improved; the access to the posterior ends of the ribs is simplified, and it is not necessary to carry the incision quite so high as when the muscles are cut through and not reflected.

4. The two suture lines are not superimposed, and if the skin sutures should give way, the extrafascial space is protected by the muscle layer.

5. There is virtually no bleeding from the muscle layer at the second stage.

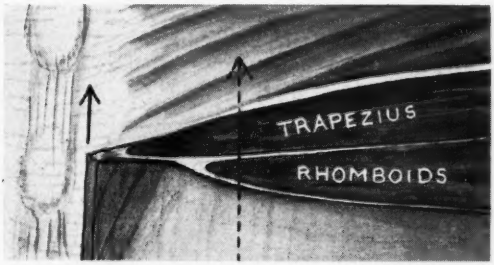

Fig. 3.—Drawing of a section through the trapezius-rhomboid layer at the level of the fifth thoracic spinous process. The broken line indicates the usual site of division of the muscles. The unbroken arrow shows the proposed line of division.

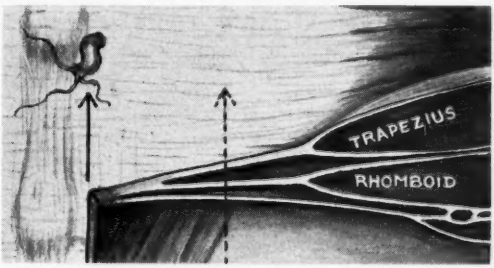

Fig. 4.—Drawing of a section through the trapezius-rhomboid layer at the level of the second thoracic spinous process. The broken line indicates the usual site of division of the muscles. The unbroken arrow shows the proposed line of division. Note the position of the posterior scapular artery deep to the rhomboid muscle; in this example it is well out of the way of either incision.

This freeing and displacement of the trapezius-rhomboid muscle mass, as opposed to its deliberate division, is somewhat comparable to the treatment of the rectus muscle in a muscle-displacing paramedian incision as opposed to the older incision that is carried straight down through the rectus sheath and rectus muscle, separating and paralyzing a medial strip of the rectus. I feel that many surgeons may wish to use it in posterior thoracoplasty in preference to what is, I think, a cruder and less desirable technique.

TREATMENT OF SYPHILITIC ANEURYSMS BY CELLOPHANE WRAPPING

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THE surgical treatment of aneurysms still is an open field despite the various methods mentioned in the medical literature and even despite the modern surgical techniques available at the present time. According to Pearse,[1] the surgical attempt at producing progressive obliteration of the lumen of the great vessels appeared in the nineteenth century. Even before Moore in 1864 tried the wiring technique, small particles of metal were inserted into the aneurysm sac to encourage thrombosis. It soon became apparent that small, loose foreign bodies introduced into the aneurysm sac frequently escaped into the circulation, causing immediate death. The wiring of aneurysms then became popular and is still occasionally used. This consists of inserting from 30 to 200 feet of fine silver or copper wire into the aneurysm sac. A galvanic current was sometimes passed through the wire to encourage thrombus formation.

Dr. J. M. T. Finney, of Johns Hopkins Hospital, according to his personal communication to Emmert,[2] wired thirty-five aneurysms, with definite improvement reported in 50 per cent and no change in the other 50 per cent. It is interesting to note that while Finney reported that 50 per cent of the patients treated by wiring showed definite improvement, Babcock[3] mentioned statistics on thirty-nine patients treated by the same process at the Johns Hopkins Hospital, nearly 50 per cent of whom died in from one day to two months after wiring, and none of whom were known to be living at the time of Reed's report in 1926. Wheeler[4] reported on three patients operated upon in 1910, 1917, and 1928, in whom a whisp or a cage of Colt's wire was used; two patients survived four and eight years and one died three months after the operation from leakage of blood around the point of the wire into the aneurysm wall. This represents one of the most common and fatal complications following the wiring procedure. Even at the height of its popularity, the effectiveness of the wiring technique was recognized as limited to the saccular type of aneurysm, which is much less frequent than the fusiform type.

The anastomosis between the common carotid artery and the internal jugular vein is a method of treatment of aneurysms of the aorta advised by Babcock[3] but never very widely adopted. His procedure provides a by-pass which permits the blood of the aorta to escape to the lower negative pressure system of the vena cava.

A number of methods and devices have been advocated for producing occlusion of large blood vessels, with unsatisfactory results in many instances. In addition to the ineffectual results and fatal complications accompanying the

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use of many of these methods, almost all of them, with one or two exceptions, are too traumatic for application to a dilated, thin-walled syphilitic aneurysm which is about to rupture spontaneously. These methods of occlusion may be classified under three headings as follows: (1) insertion of foreign bodies into the lumen of the vessels to produce thrombosis; (2) external compression of the vessel; (3) injuries to the wall of the vessel, resulting in fibrosis and constriction of the lumen.

The results of insertion of foreign bodies into the lumen of the vessels have already been described as applied to the wiring of aneurysms. The method is applicable only to saccular aneurysms in which a current of blood is sufficiently sluggish to permit the development of thrombosis. It also has an appreciable mortality from leakage about the point of insertion of the wire.

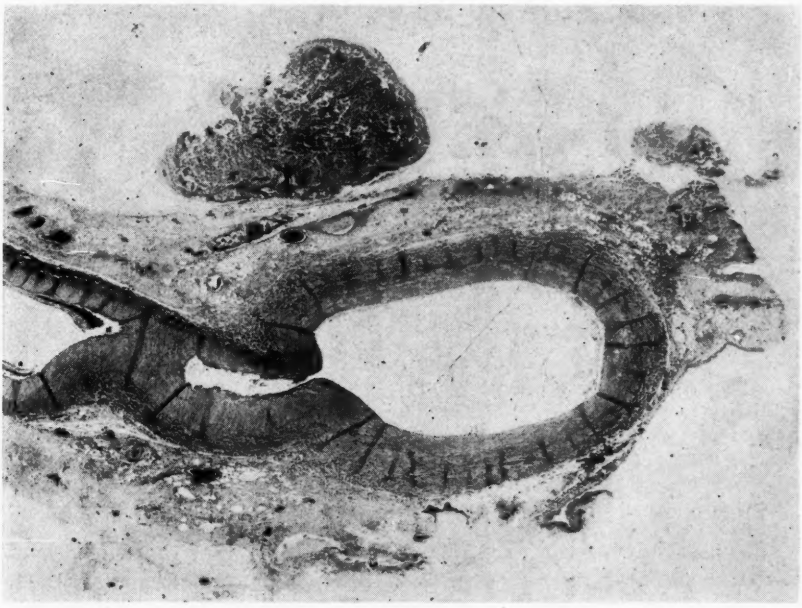

Fig. 1.—Cross section of aorta of dog wrapped for two weeks with physiologically inert 100 P.V. 000 type of cellophane. This section represents an almost normal aorta since almost no fibrous tissue reaction is present.

External compression of the wall of the vessel has been arranged in various forms to provide progressively increasing pressure by means of a spring device or an adjustable clamp regulated from outside the skin. Examples of this method are provided by the rubber tube of Matas, the Neff or Halsted clamp, the fascial strip and the Matas band. The disadvantages of this method are twofold: first, the pressure atrophy and consequent disruption of the vessel and, second, the tendency for infection to enter through the sinus tract.[5]

Most of the methods advocated for causing fibrosis and constriction by injury to the wall of the vessel are entirely too traumatic for consideration in

the case of an aneurysm in addition to proving ineffectual in reducing the size of the lumen of a normal vessel. The injection of sclerosing solutions and silica into the wall of the vessel, the painting of irritating solutions on the outside of the vessel, the use of plication sutures in the vessel walls and cauterization of the vessel wall have all failed to produce permanent constriction of the lumen when applied to normal vessels, according to the work of Pearse in 1940. The same work showed that the chronic progressive irritation, fibrosis and foreign body reaction caused by the insertion of ordinary 300 P.T. cellophane in close proximity to the vessel provide a method of thickening the wall and occluding the lumen of a blood vessel without the objections mentioned.

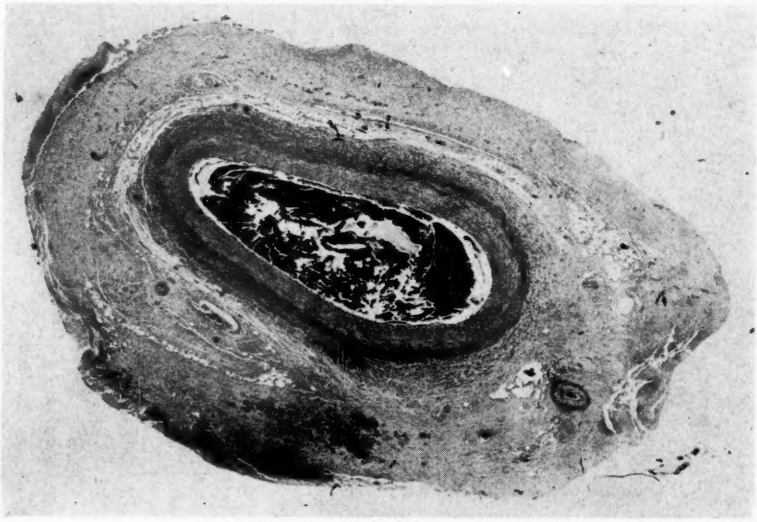

Fig. 2.—Cross section of aorta of same dog as illustrated in Fig. 1 except that this area had been wrapped for two weeks with physiologically reactive 1.5 mil polythene type of cellophane. Note marked increase in thickness of wall and decrease in size of lumen.

The first recognition of the irritating properties of cellophane resulted from the studies of Page,[6] who wrapped the kidneys of dogs in cellophane in 1939 to produce hypertension. A marked tissue reaction was said to have occurred within three to five days following the application of this cellophane.[7] Within two weeks a dense fibroblastic and collagenous deposit developed over the surface of the kidney which formed a constricting hull from 3 to 5 mm. in thickness. This hull continued to thicken for an undetermined period of time despite the disappearance of the cellophane.[6] A foreign body reaction was said to have been produced by all of the varieties of cellophane used, although the moisture-proof types appeared less reliable than the others. Other organs such as the heart, liver, and adrenal glands were also wrapped in cellophane, but no final results were reported.[8]

Harrison and Chandy[9] reported on two patients with aneurysm of the subclavian artery who were treated by cellophane fibrosis. The aneurysm dilata-

tions were found to be completely reduced within fifteen months in one of these patients, with complete interruption of the artery by a ring of fibrous tissue four months later. Harper and Robinson[10] occluded a patent ductus arteriosus with 300 P.T. cellophane, around which two No. 8 braided silk ligatures were tied. The murmur disappeared immediately after the operation, but became audible

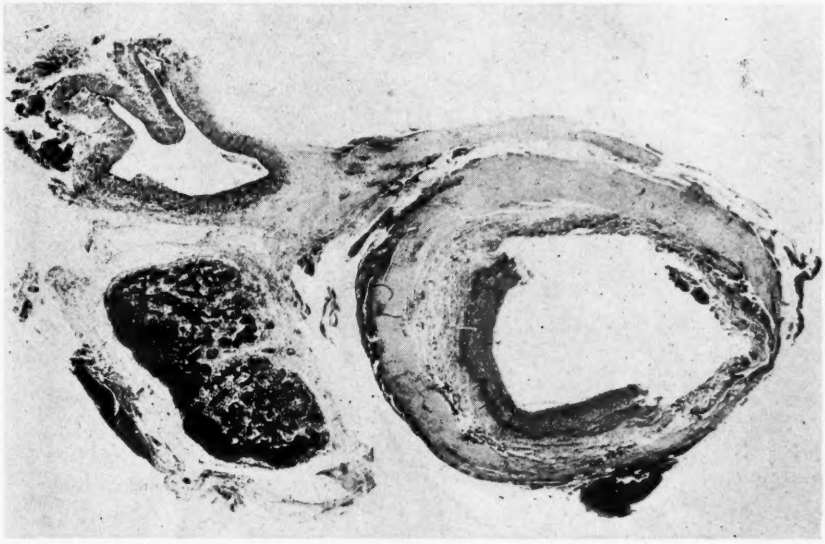

Fig. 3.—Cross section of aorta wrapped for one month with 100 P.V. 000 cellophane representing a normal control area.

Fig. 4.—Cross section of aorta of same dog illustrated in Fig. 3 except that this area was wrapped with 1.5 mil polythene cellophane for one month.

Fig. 5.—Two cross sections of aorta, lower right one of which was wrapped in polythene cellophane for two and one-half months. Note marked thickening of wall and decreased diameter of lumen in comparison with untreated control area represen.ed in left upper corner.

Fig. 6.—A section of thickened wall of aorta shown in Fig. 5 under higher magnification, illustrating fibrous nature of the reaction.

again two weeks later. A marked diminution of the murmur was noted two months later and complete disappearance, two and one-half months after its recurrence. No further recurrence of the murmur was noted in the one and one-half years during which the patient was followed. Similar reports are occurring more and more frequently in the medical literature. However, some dissatisfaction has been noted among some other surgeons who have used cellophane in attempting to obliterate the vessel lumen in cases of patent ductus arteriosus.

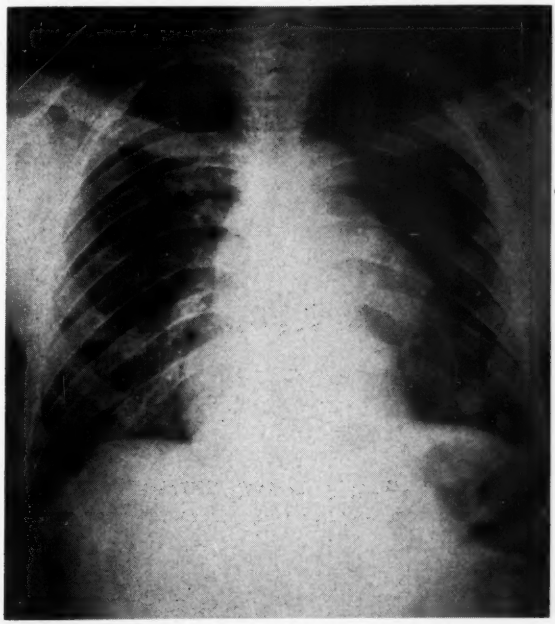

Fig. 7.—Roentgenogram of chest, posteroanterior view, illustrating fusiform aneurysm of descending aorta which was wrapped with polythene cellophane.

In direct contradistinction to the work of Page, Pearse, and Harrison and associates are the results of another group who considered cellophane as a physiologically inert substance suitable for application and insertion into areas of the body in which a foreign body reaction and adhesions would be very undesirable. Donati[11] was the first to demonstrate this inert property of cellophane in 1936. Regular commercial cellophane, .02 mm. in thickness, was used to prevent adhesions between the brain and skull, with evidence of no greater irritation than that caused by other materials. The cellophane was found close to the brain after thirty-five days with no irritative reaction surrounding it. Later, McKeever[12] used cellophane in the treatment of arthritis. He used type 300 P.U.T. 71 and concluded, as did Donati, that no reaction is caused by cellophane for at least two months after operation. This impression was confirmed by microscopic studies of a section of tissue taken from the vicinity of the cellophane in the injured knee.

Wheeldon[13] performed approximately fifty operations of arthroplasty, using 300 moisture-proof cellophane as the lining of joints. Again he agreed with McKeever, stating, "In this series of cases there has been no irritation of tissues by cellophane in any way." He had also used the material as a nonabsorbable suture and believed that irritation did not occur immediately after operation, or at any subsequent time.

Fig. 8.—Roentgenogram, left lateral view, of chest shown in Fig. 7.

These various physiologic reactions of cellophane exhibited diametrically opposite properties. On one hand, cellophane represented an inert substance producing practically no foreign body reaction when left in the tissue and, on the other hand, inducing a violent fibrous tissue reaction resulting in fibrosis and contraction. This led to the conclusion that different varieties of cellophane produced entirely different physiologic reactions, depending upon the chemical structure of the material. Before any satisfactory clinical application could be made of the properties of cellophane to produce a constrictive fibrosis, a distinction was needed between the physiologically inactive and reactive types of cellophane. For that reason, a series of animal experiments was conducted on dogs, in which five different basic types of cellophane were used.

An investigation of different types of transparent films* revealed that only a few chemically different basic types are used. The large number of varieties

*Supplied through the courtesy of the E. I. du Pont de Nemours and Company, Cellophane Division, Wilmington, Del.

is produced by combinations of these basic types with a wide variety of coatings to satisfy the packaging or other functional requirements of various industries. These coatings provide such quality characteristics as resistance to moisture vapor transmission in various degrees, heat sealing, packaging machine running qualities, etc. Some of these coatings resist the action of free water, whereas others do not. For these reasons, basic types of cellophane, or other films, have been used in most of these experiments with the results presented in Table I.

TABLE I

TYPE OF FILM	CHARACTERISTICS	PHYSIOLOGIC REACTION
Cellophane 300 P.U.T. 71 300 P.T. 62	Cellulose hydrate; nonmoisture proof; insoluble in water and dilute acids	Little reaction
P.V. film 100B P.V. 000	Polyvinyl alcohol; water soluble	Little reaction; technical difficulties due to solubility of film
Polythene 1.5 mil	Insoluble in water and dilute acids	Marked fibrous reaction
Cellulose acetate 250 C.A. 40	Highly purified cellulose acetate film; insoluble in water and dilute acids	Little reaction

EXPERIMENTAL TECHNIQUE ON DOGS

Under nembutal anesthesia, a long, left pararectus incision was made from the costal margin downward. The abdominal aorta was exposed and freed throughout its entire length, preserving its major branches. Strips of double thickness cellophane of different varieties, each about one-half inch in width, were then passed around different sections of the aorta. Only two or three different strips were used on any one animal to provide a wide space between the different varieties of cellophane and to avoid overlapping of reactions. These different types of cellophane were carefully diagrammed as to location on each animal to assure accurate interpretation of the reaction at autopsy. A major branch of the abdominal aorta was used to separate the different strips to prevent their slipping together. The two ends of each strip were sutured together with fine silk to hold the ring of cellophane loosely around the aorta. The peritoneum was then closed loosely over the aorta and retroperitoneal space and the abdominal wall was closed.

Comments.—The abdominal aorta was used in preference to the thoracic aorta to eliminate the complications involved in providing positive intratracheal pressure to avoid pulmonary collapse. An unsuccessful attempt was made to produce sclerosis by painting the aorta with rose aniline dye similar to that used in the manufacture of indelible pencils, which is known to have produced a marked fibrotic reaction. This proved fatal due to the production of extensive necrosis of the liver and spleen. The period of observation in two of the animals was shortened by the development of an infection in one, apparently due to contamination at operation, and by distemper in another.

Results.—A very marked fibrous tissue reaction surrounding the section of aorta in close proximity to the polythene variety of cellophane was noted in

both animals in which it was used. The reaction was still acute as indicated by the cellular proliferation in the first animal in which the tissue was examined two weeks postoperatively. A very dense fibrous proliferation was present in the tissue surrounding the section of aorta covered with the polythene variety of cellophane in another animal sacrificed two and one-half months postoperatively. In this case, not only had the wall of the aorta increased to six times its normal thickness, but the lumen of the vessel had been reduced to about one-third of its normal diameter.

Following is a report of the successful clinical application of the polythene variety of cellophane to a fusiform aneurysm of the thoracic aorta:

CASE REPORT

W. W. (History No. 1222248), a 36-year-old colored man, was admitted on the Chest Service of the Barnes Hospital with a history of syphilis, first discovered by means of a positive Wassermann in 1927. Following this, he was given eighteen hip and eighteen arm "shots," which constituted his entire course of antisyphilitic therapy. The patient began to complain of a dull aching pain in the mid-back, slightly to the left of the midline, in June, 1944. This pain became progressively more severe and was not affected by a change in position. The pain was noticeably increased by coughing, sneezing and straining. A throbbing ache was also noted in the left side of the anterior part of the chest during the previous four months. The patient had no complaints of cough, dyspnea, palpitation, syncope or hoarseness. A moderately large, pulsating aneurysm was discovered immediately below the arch of the aorta in the left posterior mediastinum in February, 1945, at which time the patient refused to have it treated surgically. Five months later he returned with a definite increase in his symptoms and requested surgical treatment. At this time a slight enlargement was noted in the size of the aortic aneurysm, although there was no definite x-ray evidence of its erosion into a vertebra. The blood pressure on the right was 98/66 and on the left, 104/70. No murmurs or thrills were heard over the patient's chest. On July 6, 1945, an exploratory thoracotomy was performed through a posterolateral incision with the resection of the sixth rib. A large, pulsating fusiform aneurysm was found involving about one-third of the descending aorta, centering opposite the seventh vertebra. The aorta in this area was enlarged to about three times its normal size and was loosely adherent to the lung, which was carefully peeled away by blunt gauze dissection. The mediastinal pleura was also excised from the aneurysm and the aneurysmal sac exposed over about 80 per cent of its surface. The posterior aspect could not be reached due to its beginning erosion into the bodies of the vertebrae and adjacent ribs. A single layer of 1.5 mil polythene cellophane was tacked loosely over the exposed areas of the aneurysm by means of about six fine silk sutures placed in the wall of the aorta above and below the aneurysm and in the adjacent structures. The left lung was then re-expanded, the chest wall closed and the excess air aspirated from the left pleural cavity. The postoperative course was essentially uneventful with a return of the patient's temperature to normal on the fifth postoperative day following a thoracentesis with the removal of 300 c.c. of serosanguineous fluid from the left pleural cavity. At the time of his discharge on the thirteenth postoperative day, the patient was ambulatory and completely afebrile, complaining only of a continued pain in the left side of the posterior part of the chest. This pain has gradually decreased, and the throbbing ache in the left side of the anterior part of the chest completely disappeared during the first three months after his operation. Admittedly, a longer period of observation will be needed to determine the late results of such treatment.

The patient was admitted July 3, 1945, and discharged July 19, 1945.

Three other syphilitic aneurysms of the ascending aorta have also been wrapped with polythene film, but insufficient time has elapsed to provide any data on the constrictive reaction. One aneurysm was about ready to rupture

at a thin, soft spot on its anterior wall where it rubbed against the posterior surface of the sternum with each pulsation and respiratory cycle. These other aneurysms extended to the right and caused marked distortion of the surrounding structures, particularly the superior vena cava and trachea. These structures required separation from the distorted aorta following incision of the mediastinal pleura. The sheet of cellophane was then slipped under the superior vena cava and the mediastinal pleura was drawn between the cellophane and vena cava to separate them and reduce the effect of the cellophane on the vena cava. Some difficulty was occasionally encountered in procuring sufficient mediastinal pleura to completely cover the cellophane. The most satisfactory way to avoid the contact of the surrounding structures with this extremely reactive polythene film seems to be to cover it with a layer of less reactive cellophane, such as 300 P.U.T. 71, 300 P.T. 62, or one of the moisture-resistant varieties. Further animal experiments are being carried out at the present time on refinements in technique, this representing a preliminary report.

Polythene film was also used recently for the ligation of a recanulated ductus arteriosus in a 14-year-old colored girl with subacute bacterial endocarditis in whom the patent ductus had been ligated four years previously with cotton umbilical tape. Any attempt at transection of this poorly accessible Botallo's ductus, which was firmly bound down in scar tissue, would have been impossible and fatal. The use of a reactive type of cellophane in this case provided a highly satisfactory solution to a difficult problem. This would seem to present a satisfactory method of treating all patent ducti and a much less dangerous one than transecting such short and broad vessels between clamps. A certain amount of caution is necessary, however, to keep the cellophane away from the recurrent laryngeal nerve.

CONCLUSIONS

1. The use of polythene cellophane seems to offer a successful method of gradual fibrosis and obliteration of aneurysms and large vessels.

2. Experimental studies reported here have shown that in order to obtain the desired result, it is necessary to use a particular type of cellophane, since some varieties produce no fibrosis.

3. Polythene cellophane has been found useful not only in the treatment of aortic aneurysms, but also in the obliteration of a patent ductus arteriosus.

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CYSTIC DISEASE OF THE LUNGS AND ITS RELATIONSHIP TO BRONCHIECTATIC CAVITIES

A Study of Twenty-Two Cases

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A WIDE variety of names has been given to the condition commonly known as cystic disease of the lungs, These various terms indicate the different views which have been held as to the genesis of the cysts found in the lungs. Some of the more common names are pulmonary cysts, bronchogenic cysts, congenital cystic disease of the lungs, pneumocysts, honeycomb lung, solitary and balloon cysts of the lungs and pneumatoceles.

The term cystic disease of the lung, it seems to us, can be used for all abnormalities of pulmonary tissue in which cysts or cystlike structures are found, regardless of pathogenesis. It includes "cystic" bronchiectasis which refers to the rather large cystlike cavities sometimes produced by bronchiectasis but not to the ordinary cylindrical and saccular forms of bronchiectasis, mediastinal cysts of bronchiogenic type, nor to such specific entities as parasitic or hydatid cysts, tuberculous cavities, ordinary pulmonary abscesses with little or no attempt at epithelization of cavities or cavitary bronchogenic carcinoma. By broad definition, all pulmonary cysts or cystlike cavities may be considered forms of bronchiectasis; however, cystic disease of the lungs has commonly been considered a separate condition and is so regarded in this paper.

In this paper, too, the term congenital bronchiectasis has been avoided in connection with the cysts included under cystic disease of the lungs because it seems to us that its use has a tendency to confuse the issue. In our present state of knowledge, it is probably better to use the term congenital bronchiectasis only for what is believed to be the rare forms of bronchiectasis, which presumably are present from birth.

HISTORICAL ASPECTS AND REVIEW OF THE LITERATURE

Cystic disease of the lungs undoubtedly existed for many years without being recognized. As far as can be determined, no reports of pulmonary cysts, as such, appeared prior to the seventeenth century. The earliest report of cystic lungs was probably that by Thomas Bartholinus; a translation of his cases from the Leyden edition of Malpighius, in 1687, may be found in the article of recent years by Oughterson and Taffel.[1]

One of the best of the early twentieth century American descriptions of this condition was that by Koontz[2] in 1925, who was able to collect 108 cases

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from the world literature. He reviewed the findings at necropsy as reported in the literature and reported a case of his own. In the report of this case he included a detailed description of the pathologic changes.

Since 1925, the number of reports of cases of cystic disease of the lungs has increased greatly both in America and Great Britain. Among American writers, Wood and others,[3-7] Schenck[8, 9] and Maier[10, 11] recently have been outstanding contributors to knowledge of this condition. Among the British authors of recent times, Smith,[12] Sellors,[13] Willis[14] and Willis and Almeyda[15] have been prominent in the investigation of this condition.

Although cystic disease of the lungs is considered rare, Schenck,[8] in 1937, stated that a total of 381 cases of cystic disease of the lungs had been reported since the original description. Wood,[6] in August of the same year, added forty-eight cases in which the patients were seen at the Mayo Clinic. Within recent years, many excellent reports have appeared and the condition is being diagnosed with increased frequency and accuracy.

CLASSIFICATION OF CYSTIC DISEASE OF THE LUNGS

The following working classification is offered for cysts found in cystic disease of the lungs:

I. The true developmental or so-called congenital cysts of the lungs (some of the bronchogenic cysts or bronchoalveolar cysts)

II. Acquired cysts or cystlike cavities of the lungs
   - A. "Cystic" bronchiectasis
   - B. Other pulmonary cysts or cystlike cavities
     1. Pneumatoceles
     2. Abscess cysts
     3. All other pulmonary cysts, including some bronchogenic cysts (these pulmonary cysts predominantly result from infection, trauma [penetrating wounds of chest], noxious gases, hemorrhagic effusions and other contributory factors or combination of factors)
     4. Emphysematous blebs
     5. Emphysematous bullae

True developmental or so-called congenital cysts of the lungs are an important type of cyst. In addition to being located in the pulmonary parenchyma, they sometimes are found near or attached to the bifurcation of the trachea or to a main stem bronchus. The essential histologic feature of this type of cyst lies in the uniformity of the lining membrane which maintains the regular character of the bronchial epithelium (Fig. 1). In contrast to the regularity of arrangement of the bronchial epithelium is the disorderly arrangement and the erratic grouping of the supporting structures of the wall which is of the bronchial type. Cartilage, smooth muscle, elastic tissue and smooth glands are arranged irregularly. This arrangement is in marked contrast to

the orderly arrangement of these structures associated with simple dilatation of a bronchus. An extreme variability in location and number of these features is almost characteristic of the developmental cyst. One or another of the stromal elements in these developmental cysts may be absent, or one of these elements may be prominent or even hyperplastic and, in a few instances, a picture almost like that of true neoplasia may result.

Of the acquired cysts, "cystic" bronchiectasis will be considered in detail in Table I, and the only other type which needs special mention is the pneumatocele. The pneumatocele has no epithelial lining and therefore may subside spontaneously. The distinction between "cystic" bronchiectasis and other acquired and congenital pulmonary cysts is difficult. The criteria given in Table I are of value in the histopathologic differentiation of "cystic" bronchiectasis from other cysts included under cystic disease of the lungs, especially the cysts of developmental or congenital origin.

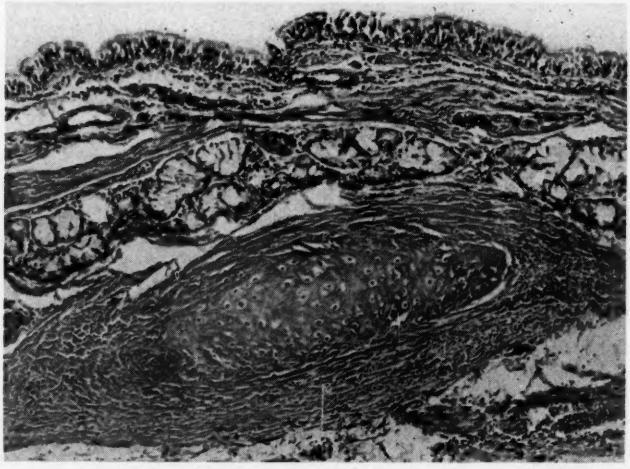

Fig. 1.—Portion of cyst wall shows the bronchial type of lining. The numerous mucous glands may be seen, as well as a portion of cartilage beneath the mucous glands (hematoxylin and eosin ×85).

STUDY OF TWENTY-TWO CASES

Twenty-two surgically treated patients with cystic disease of the lungs from whom tissue has been removed at the Mayo Clinic were selected for this study. No cysts of the mediastinum were included in this study although developmental or so-called congenital cysts of bronchial type do occur in the mediastinum. Each case was reviewed from the clinical standpoint and an analysis was made of the outstanding features. A detailed study also was made of the histopathologic features. Blocks were cut from the walls of each cyst and sections were prepared for microscopic study with hematoxylin and eosin stains in all cases; special stains, such as van Gieson's and mucicarmine, were used in selected cases. The character of the cyst lining was noted particularly. In addition,

TABLE I. COMPARISON OF PATHOLOGIC FEATURES OF "CYSTIC" BRONCHIECTASIS AND ALL OTHER TYPES OF CYSTIC DISEASE OF THE LUNG

	"CYSTIC" BRONCHIECTASIS	CYSTIC DISEASE OF THE LUNGS INCLUDING CONGENITAL OR DEVELOPMENTAL CYSTS
Size of cysts	Usually small, measuring from 2.0 to 3.5 or 4 cm. in diameter, but may be larger	Usually measures more than from 3 to 4 cm. in diameter and may reach huge dimensions
Location of cysts	Most frequently but not always in basilar lobes	Any place in lung or even in aberrant or accessory pulmonary tissue within thorax
Type of bronchial communication	Bronchi always involved and communicating with process; always enter cystic cavity directly	Bronchi may or may not enter cyst directly; bronchial communication may be small and imperfect but is usually present (rarely absent) and may be found on careful gross or microscopic examination (sometimes serial sections are needed to show it)
Usual relation of cysts to bronchi	Cystlike cavities are part of bronchiectatic process and are concentric to involved bronchi	Cysts are not part of a bronchiectatic process but bronchiectasis may develop secondarily; cysts are placed eccentrically in relation to bronchi
State of communicating or associated bronchi	Communicating bronchi always show dilatation and thickening of wall	Communicating bronchi may be normal, dilated or contracted
Condition of pulmonary parenchyma surrounding cysts	Usually considerable chronic pneumonitis with or without atelectasis	Not infrequently normal or minimal inflammatory process but occasionally chronic pneumonitis; atelectasis is often present about cyst
Microscopic findings in cyst cavities	Marked inflammatory process with irregular roughened lining; lining epithelium may be lost or ulcerated but there is a remarkable retention of epithelial lining; stromal elements show a regular arrangement or pattern	Lining epithelium is usually of bronchial type (stratified columnar epithelium with cilia present); in majority, bronchial elements in stroma are arranged erratically and these frequently show hyperplasia (sometimes to a considerable degree); some forms of cyst may have no epithelial lining and wall may be thick, fibrous and very inflammatory

the presence with the relative amount or absence of the following features was given consideration: inflammatory reaction, cartilage, mucous glands, lymphoid tissue, smooth muscle, elastic or fibrous connective tissue, vascularity, pigment, giant cells and the alveolar tissue.

The cystic disease of the lungs in these twenty-two patients could be classified into three groups as follows: developmental or congenital cysts in twelve, acquired cysts (including "cystic" bronchiectasis) in six and indeterminate cysts in four.

The final classification was arrived at by giving precedence to that feature of the case which seemed to be predominant. The largest group of cases was classed among the so-called congenital or developmental cysts. All of the cysts of this type had a definite bronchial type of lining. In the four patients in whom the cyst was of an indeterminate type, the lining was of an indeterminate type or simply fibrous in character. Also the elements of the stroma within the cyst wall did not suggest bronchogenic origin so that the condition in the four patients was classified in this way by necessity.

A summary of some of the important pathologic features in the twenty-two cases of pulmonary cysts may be seen by referring to Table II. Most of the cysts had a bronchial communication. Infection was present within the cyst in all

TABLE II. SOME OF THE FEATURES IN TWENTY-TWO SURGICAL CASES OF CYSTIC DISEASE OF THE LUNGS

CASE	AGE (YR.)	LOCATION OF CYST WITHIN LUNG	EXTENT OF INVOLVEMENT	BRONCHIAL COMMUNICATION	CONTENTS OF CYST	LINING OF CYST	TENTATIVE CLASSIFICATION OF TYPE OF CYST
1	38	Azygos lobe	From one-half to two-thirds of right upper hemithorax	Yes	Pus	Bronchial type	Developmental
2*	38	Right upper lobe	Upper one-third of right hemithorax	No	Thick, brown fluid	Bronchial type	Developmental
3	21	Left lower lobe	Limited to left lower lobe	Yes	Mucopus	Bronchial type	Developmental and acquired†
4	34	Left hemithorax	Practically all of left hemithorax	No	Chocolate-colored, thick material	Flat-celled indeterminate type	Indeterminate
5	12	Right upper and middle lobes	Right upper and middle lobes	No	Mucopus	Bronchial type	Developmental
6	26	Right lower lobe	Portion of right lower lobe	No	Mucopus	Bronchial type and squamous epithelium	Developmental and acquired†
7	47	Right lower lobe	Right lower lobe	No	Pus	Bronchial type and squamous epithelium	Developmental and acquired†
8	7	Left upper lobe	One-half of left upper lobe	Yes	Mucopus	Indeterminate type	Indeterminate
9*	13	Azygos lobe	Portion of right upper hemithorax	Yes	Mucoid material	Bronchial type	Developmental
10	25	Right upper and middle lobes	Right upper and middle lobes	Yes	Pus	Bronchial type	Developmental
11	26	Left upper lobe	Left upper lobe	Yes	Dark, clotted blood	Fibrous inflammatory wall	Indeterminate
12	30	Right lower lobe	Most of right lower lobe	No	Mucoid material	Bronchial type	Developmental
13*	27	Right upper and middle lobes	Right upper lobe and portion of middle lobes	Yes	Hemorrhagic mucopus	Bronchial type	Developmental
14	28	Upper one-third of left hemithorax	Upper one-third of left hemithorax	No	Mucoid material	Indeterminate type with flat cells	Indeterminate
15	18	Right lower lobe	Right lower lobe	No	Mucopus	Bronchial type	Developmental
16	43	Left lung	Most of left lung	Yes	Mucopus	Bronchial type and squamous epithelium	Developmental and acquired†
17	39	Right upper and middle lobes	Greater portion of right upper and middle lobes	Yes	Mucopus	Bronchial type	Acquired
18	36	Right lower lobe	Right lower and portion of right upper lobe	No	Mucopus	Squamous epithelium and bronchial type	Developmental
19	29	Right lower lobe	Portion of right lower lobe	Yes	Mucopus	Bronchial type	Acquired
20	45	Left lower lobe peripherally	Not significant	No	Thin, cloudy fluid	Fibrous inflammatory wall	Acquired
21	15	Left upper lobe	Left upper lobe	Yes	Mucopus	Bronchial type	Acquired
22	20	Right upper and lower lobes	Dorsal portion of right lower and small portion of right upper lobe	Yes	Mucopus	Fibrous inflammatory wall with ulceration of bronchial epithelium	Acquired

*No infection within the cyst.

†Developmental features predominant; therefore considered in text as developmental cysts.

but three cases. In none of the twenty-two patients in this series was there any evidence that bronchogenic carcinoma had its origin within the wall of one of these benign cysts.

One case has been selected as being fairly illustrative of all clinical and surgical features in the twenty-two cases of the pulmonary cysts and is presented in detail.

CASE 18 (Table II).—A woman, 36 years of age, complained of cough, productive of thick, purulent sputum, hemoptysis and frequent colds. These symptoms had been present for six years. Onset had followed pneumonia. The patient was in bed for eleven months at the time of the first attack and had had several pulmonary hemorrhages since the initial onset. Her disability because of symptoms previously mentioned and fatigue was considerable. Physical examination at the clinic revealed elevation of the right side of the diaphragm and decreased breath sounds over the base of the right lung. Multiple roentgenologic examinations

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Fig. 2.—Large pulmonary cyst occupying the greater portion of the right lower lobe of the lung.

revealed a tumor of the lower lobe of the right lung posteriorly, which apparently was connected to the mediastinum. The roentgenologist considered the possibility of a bronchogenic cyst. The sputum was repeatedly negative for tubercle bacilli. Bronchoscopy was performed, at which time it was noted that pus was coming from the apical branch of the bronchus of the right lower lobe. Operation was recommended because of the failure of conservative measures to give relief. A primary right posterolateral incision was made May 1, 1944, and a long segment of the sixth rib was resected. A large cyst was palpable in the upper portion of the lower lobe. The inflammatory reaction was marked around the cyst and extended into the adjacent portion of the upper lobe. An attempt was made to establish a

line of cleavage between the upper and lower lobes but, in doing so, the cyst was ruptured and considerable contamination resulted. The cyst was large, measuring about 8 or 9 cm. in diameter, and contained some foul debris. Dissection and individual ligation of the hilar structures were carried out and the right lower lobe was removed. Closed drainage was instituted. Bronchoscopy was performed at the end of the operation. The operative diagnosis was infected cyst of the right lung.

Gross examination of the tissue removed (Fig. 2) revealed a large cyst in the lobe of the right lung. The excised right lower lobe was largely occupied by the cyst, which measured 7 by 7 by 6 cm. and was located in line with the bronchus to the right lower lobe. On gross examination no communication was found between the bronchi and the cyst. The bronchial radicles were compressed by the cyst. The lining of the cyst was rather smooth and the surrounding pulmonary tissue was atelectatic. On microscopic examination the cyst was lined with pseudostratified columnar epithelium of the bronchial type, with a ciliated border in some places, but a large portion of the sections showed a squamous type of epithelial lining. Considerable inflammation was present in the wall of the cyst with ulceration of the lining. Cartilage and smooth muscle were noted in the wall of the cyst. Mucous glands were numerous and fibrous connective tissue was a prominent feature of the stroma within the wall. A small amount of lymphoid tissue was present. Small and large blood vessels were abundant throughout the wall of the cyst. Anthracotic pigment was noted within the pulmonary parenchyma surrounding the cyst. The parenchyma also showed chronic pneumonitis. After operation, secondary anemia and empyema developed. The empyema subsided after open operation by one of us (O. T. C.). Follow-up examination revealed that the surgical result was satisfactory.

COMMENT

From this study there appear to be many factors in the etiology and pathogenesis of cystic disease of the lungs which may be impossible to determine in a given case with any degree of certainty. Developmental factors, infection with its sequelae, mechanical and physiologic factors are all concerned as sources of origin of these pulmonary cysts. It appears unlikely that these cysts can be classified with assurance as being totally congenital (developmental) or totally acquired. For example, the so-called congenital cysts may be influenced profoundly by acquired factors and a cyst that is thought to be acquired may be influenced a great deal by the presence of a malformation or developmental anomaly. In our present state of knowledge, it does not seem possible to state whether a small, actual or potential cyst may exist at birth and may be detected for the first time later in life. Some developmental or congenital cysts have been reported as developing during life and, on comparison with previous roentgenograms, were not present earlier in life. The acquired nature, however, may have been only apparent and not actual. On the other hand, some pulmonary cysts do appear to be predominantly acquired. The large size of the pulmonary cyst, the eccentric position of the cyst in relation to communicating bronchi, normal communicating bronchi, a bronchial wall with erratic arrangements of stromal elements and hyperplasia of the latter suggest a developmental or congenital origin. The developmental or so-called congenital origin is more certain in cysts found in accessory or aberrant pulmonary tissue, or in mediastinal cysts with a bronchial type of wall.

Cystic disease of the lungs may be extremely variable in regard to the size, location and the resulting complications of the cysts. The pathologic picture at operation may be simple and uncomplicated, or it may be bizarre in the

presence of hemorrhage and infection within the cyst and their resulting complications. The presence of pulmonary cysts and their complications may profoundly alter intrathoracic physiology. Accessory or aberrant lobes of the lungs may be associated with anomalous vessels and both are occasionally encountered in conjunction with cystic disease of the lungs. The bronchial communication present in cystic disease frequently results in the patient's coming to the attention of the surgeon.

Diagnosis.—The symptoms of cystic disease of the lungs are protean and are largely dependent on the size of the cyst, its location and the presence or absence of a bronchial communication. Hemoptysis or even severe pulmonary hemorrhage is not an uncommon symptom in cystic disease of the lungs. Hemoptysis and even severe hemorrhages were present in sixteen of the twenty-two patients in this series. A multitude of complications occurring with cystic disease of the lungs may simulate most any condition found in the chest. Pulmonary tuberculosis and bronchogenic carcinoma may coexist with cystic disease of the lungs. Pulmonary tuberculosis is a diagnosis which has frequently been made in these cases; the condition of a number of patients in our group had been so diagnosed and they had even spent long periods in sanatoria.

An accurate diagnosis of cystic disease of the lungs is largely dependent upon adequate roentgenographic examination of the thorax. It is felt that bronchoscopy is of definite value, especially for the patient who is to be treated surgically, to rule out any possibility of other bronchial lesions. Occasionally, an accurate diagnosis can be arrived at only by exploratory thoracotomy.

In the differential diagnosis, consideration must be given to almost all intrathoracic neoplasms, pulmonary abscess, localized empyema, tuberculous cavities, hydatid cysts and diaphragmatic hernias. Some eminent thoracic surgeons have admittedly treated what they thought originally was localized empyema only to discover, after failure of the cavity to become obliterated, that they were dealing with true pulmonary cysts.

Surgical Management.—Sellors[13] has pointed out how poorly the condition has been treated in the past and remarked that "the treatment of cystic disease of the lungs does not make satisfactory reading, and the number of cases that have been aspirated or drained without further attempt at cure shows how little the condition is understood."

The ideal treatment for cystic disease of the lungs is complete operative removal of the cystic portion of the lung with conservation of as much normal functioning lung as possible. Local excision is only occasionally feasible, as in a case in which the cyst may be attached by a narrow pedicle or loosely attached to the parenchyma of the lung. Most frequently it is far better to excise the entire cystic portion of lung if the distribution makes this possible and provided all other indications for such excision have been met. Drainage of the cysts and other supplementary operations, such as collapse procedures, usually are not satisfactory. Age appears to be no contraindication to surgical treatment. Operative treatment is being performed in the majority of these patients with safety, but, in some, the presence of complicating factors makes operation hazardous.

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THE MANAGEMENT OF THORACIC AND THORACOABDOMINAL WOUNDS IN THE FORWARD AREAS IN THE SICILIAN AND ITALIAN CAMPAIGNS

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THE purpose of this paper is to report the immediate results and analyze the methods employed in the management of thoracic and thoracoabdominal war wounds in the forward areas. This work was done by a surgical team of the Second Auxiliary Surgical Group during a twenty-month period, commencing with the invasion of Sicily. The employment and functions of surgical teams of an auxiliary surgical group have been described by Forsee.[1] Two hundred patients suffering intrathoracic and thoracoabdominal wounds treated in a first priority surgical hospital (a platoon of a field hospital located in physical conjunction with a division clearing station and receiving severely injured casualties who could not be transported farther to the rear, without surgery, than the rear of the division boundary) or an evacuation hospital constitute the principal basis for this paper. Sixty-four cases will be reported, in which wounds were limited to the chest wall, and the relationship of intrathoracic to spinal cord injuries will also be discussed. During the twenty months, the team functioned as a thoracic surgical team for one-half the period and as a general surgical team for the remainder. Our experience has led us to believe that intrathoracic injuries are more common than intra-abdominal wounds and that thoracoabdominal wounds constitute approximately one-quarter of all thoracic injuries with pleural involvement. The patients treated incurred the following types of wounds as their major injury: 84 (42.0 per cent) penetrating, 61 (30.5 per cent) perforating, and 55 (27.5 per cent) thoracoabdominal. The total mortality was 9.0 per cent (18 deaths). Excluding the thoracoabdominal wounds, the mortality for the 145 intrathoracic cases was 6.2 per cent (9 deaths).

The length of time during which the patients were retained under our immediate observation is of interest because, with early surgery possible, the majority of the nontransportable patients soon became transportable. Patients with thoracic wounds were retained in the hospital an average of seven days, the shortest four days and the longest fourteen days. The thoracoabdominal patients were kept an average of ten days, the shortest eight days and the longest nineteen days. The presence of concomitant major wounds was the cause of retaining patients longer than the average periods stated here. The policy of evacuating the patients to a hospital in the base section, designated as a chest center, as soon as they were safely transportable enabled the majority to arrive in good condition and placed them in the care of experienced thoracic surgeons for any further treatment required during the optimum period of their illness. Therefore, this report emphasizes only the immediate results, and the mortality rates presented stand uncorrected for development of subsequent complications and for the lethality of concomitant wounds. It is essential, however, to note

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that 42.5 per cent of the 200 patients incurred concomitant severe injuries which often were important factors in determining the urgency of operation as well as the extent and nature of the surgery to be performed. In a subsequent report of a final follow-up to be completed, the influence of concomitant wounds and complications will be discussed. No originality is claimed for the observations or methods described in this report and proper credit cannot be given because of lack of reference facilities.

TYPES OF MISSILES

Bullets have less tearing action than do high-explosive fragments and they have two characteristics that are of interest. They cause considerable contusion to the organs through which they pass, and frequently they travel greater distances than do fragments. In bullet wounds necessitating thoracotomy, the large amount of pulmonary hematoma surrounding the tract of the missile is considerably greater than one expects from either the size of the missile or the roentgenographic findings. This has been noted repeatedly in lower lobes in thoracoabdominal wounds. Whether this is due to the wobbling of the bullet or to the high compression wave accompanying it, we do not know. The tendency of a bullet to involve more than one celomic cavity is striking. An example is a case in which a bullet entered through the left deltoid region and was found in the right iliac fossa—the patient had been lying down with his left arm extended above his head at the time of injury. Another example is that in which a bullet entered through the left clavicle and was found near the greater trochanter of the left femur.

PREOPERATIVE TREATMENT

The purpose of preoperative treatment is to restore the disturbed cardiorespiratory mechanism to as close an approximation of the normal as possible. The principles in the restoration are those involved in any seriously wounded patient to which are added certain factors peculiar to intrathoracic wounds. These are airway, chest wall pain, intravenous fluids, hemopneumothorax, and progressive tension pneumothorax.

The airway is of primary importance and the relief of pain is often essential in obtaining an open airway. In the majority of patients there is blood in the tracheobronchial tree. The amount varies from mere blood-tinged sputum to sudden massive hemoptysis. Fortunately, the latter is rare.* Encouraging the patient to cough will often suffice to clear the tracheobronchial tree. Manually holding the lower costal margin will aid in coughing. Chest wall pain may inhibit coughing and limit the depth of respiration, resulting in an inefficient hyperpnea with inadequate ventilation. This is best treated not by morphine, but by intercostal nerve block. The block should include not only the segments involved by the wound but also two segments above and below to cover the area

*An example of massive hemoptysis is that of a soldier wounded by a high explosive fragment perforating the right upper part of the chest. Shortly after admission to a field hospital, the patient had a sudden massive hemorrhage which filled the entire trachea and primary branches. Bronchoscopy cleared the trachea for the moment, but hemorrhage from the orifice of the right upper lobe bronchus continued. The hemorrhage was controlled only by packing its orifice, and the packing was held in place by also packing the entire right main bronchus. This permitted a thoracotomy to be done with suture of the bronchus and bleeding lung. The patient was seen one month later and had a fully expanded lung.

of pain overflow. If the wound is close to the vertebral column, injection of the intercostal nerves is difficult, and a paravertebral block should be done. The relief of pain will permit the patient to cough without difficulty. Clearing of the tracheobronchial tree will rapidly change the hyperpneic, cyanotic, hypotensive patient to one with deep unhurried respirations, good color, and rather prompt return of blood pressure to a more normal level. The resultant open airway, by relieving the hypoxia, will more readily raise the blood pressure than will any other procedure, except in the patients with continuing intrathoracic bleeding and progressive tension pneumothorax. Endotracheal catheter suction is imperative in clearing the airway of the unconscious patient and of the one whose cough is inadequate. In all cases of hypoxia and shock, oxygen should be given by nasal catheter. Oxygen administration should precede all preoperative measures and should be continued until the patient is ready for operation.

The position of the patient and caution in administration of intravenous fluids are important. Vital capacity is greater and coughing is more effective in the sitting position. The ideal may be approached by placing the patient in a semisitting position. However, patients with hypotension and no cyanosis or dyspnea should be placed in a Trendelenburg position. In the administration of blood, it should be remembered that a patient with partial or complete pneumothorax may develop pulmonary edema by the sudden overloading of the pulmonary vascular bed. This is especially true in patients with hypoxia due to reduction in vital capacity by mechanical means such as hemopneumothorax. The reduction in the vascular bed is a sudden one and the patients does not have time to compensate. The vascular bed is not only reduced by the partial collapse of the lung but is further reduced by the extensive hematoma of the pulmonary parenchyma which may be present. Blood replacement therapy can be given judiciously by watching for moisture in the pulmonary parenchyma. Intravenous fluids should not be started on a patient who is having difficulty coughing in the presence of gross rhonchi. The airway can be cleared, pain reduced, and thoracentesis performed in a short time, and, with relief of hypoxia, the pulmonary vascular bed will not react unfavorably to intravenous fluids.

In our experience, hemopneumothorax was so commonly encountered that treatment of its immediate effects was often an urgent requirement. The total number of hemopneumothoraces in the 200 cases was 159 (79.5 per cent). Hemopneumothorax occurred unilaterally 149 times, and bilaterally 5 times. Hemothorax alone was found in 25 cases and pneumothorax alone in 10. Neither pneumo- nor hemothorax was discernible in 4 patients. Thoracentesis was performed on the majority. This procedure assists in relieving shock by the relief of hypoxia consequent to the increased aerating parenchyma. For this reason, as much blood and air as can be obtained is removed. The largest amount was 1,100 c.c., and the average was 250 c.c. Autotransfusion has been used frequently. Occasionally the blood clots, especially if the bleeding is rapid; therefore, it is citrated as it is withdrawn and strained through gauze before being given.

The possibility of the resumption of bleeding as a result of immediate thoracentesis is frequently discussed, but is more a theoretical problem than a

practical one. The two sources of hemothorax are extrapulmonary (chest wall or mediastinal) and pulmonary hemorrhage. When resumption of bleeding does not occur it is probably due to the low pressure in the pulmonary artery and its branches. The presence of a hemothorax does not stop extrapulmonary bleeding. The objective of preoperative treatment is rapid resuscitation and thoracentesis helps accomplish it. Thus, theoretical contraindications need not be considered.

Thoracentesis was performed 100 times, and, in 73, pleural pressure readings were taken as the first step in the procedure. Pressures were recorded pre- and postoperatively and were correlated with the pre- and postoperative condition of the patient. The average positive pressure on expiration was 10 to 12 cm. of water. A few patients had positive readings on both expiration and inspiration. Following the removal of air and blood, the pressures more nearly approximated the normal. It was the exceptional patient, however, who at the end of a thoracentesis gave negative readings on both expiration and inspiration. There was not always a correlation between the degree of dyspnea and the primary pleural pressure. This was probably due to the variation in the stability of the mediastinum, with a corresponding individual variation in the transmission of transmediastinal pressures. Not all patients with positive pressures on inspiration and expiration were dyspneic, but the majority of the severely dyspneic ones presented positive pressure readings in the entire respiratory cycle.

It is of interest to note that true progressive tension pneumothorax was rare in this series. It soon became obvious, in the management of these cases, that those which required constant decompressing devices were quite rare. In only three instances was it necessary to install a decompressing catheter when the patient was first seen, and only two patients developed a true progressive tension pneumothorax postoperatively. The designation, progressive tension pneumothorax, was reserved for the patients requiring constant decompression devices. The mere fact that positive pleural readings were obtained at the first thoracentesis, in the majority of cases, makes it reasonable to classify them on the basis of treatment necessary because these patients required only intermittent thoracentesis postoperatively. We believe that most of the initial positive readings result from air from the injured lung entering the pleural cavity at the time of wounding, and the pressures may be increased from bleeding into the pleural cavity. The continued air leaks must nearly always stop soon after wounding, or remain constantly minimal, because in all cases the pulmonary involvement ranged from small penetrations or perforations to large lacerations and yet the rarity of progressive tension pneumothorax was rather striking. Perhaps the hematoma surrounding the area of the torn parenchyma creates sufficient nonaerating lung to limit the amount of air reaching the pleural cavity. The amount of pulmonary hematoma surrounding even a small tract is often considerable. Three-quarters of a lobe may be involved from the passage of a small fragment.

Immediately upon admission, the patient should be examined for sucking wounds. If one is present, it should be dressed with a large occlusive petrolatum

dressing. It is good practice to dress all wounds of the chest in this manner, for a wound labeled nonsucking may begin to suck when the position of the arm or shoulder is changed.

Roentgenograms of the chest can be obtained as soon as the patient responds to resuscitation. Erect posteroanterior and lateral views are those of choice. However, it is not always possible to have the patient sit up. If not, one must be satisfied with a prone anteroposterior view. Fluoroscopy is of value in determining the amplitude of the cardiac pulsation in suspected tamponade. It is also valuable for the few patients who do not respond to resuscitation and for whom the additional movement necessary for roentgenography is deemed inadvisable. The position of the missile can be determined in order to aid in assuming what cavities have been invaded and what structures have been involved.

Thoracoabdominal wounds require the same preoperative care, and, in addition, a Levine tube is inserted, the stomach is aspirated, and the tube is left in place. During the operation, the tube keeps the stomach deflated and aids exposure, particularly when the left transdiaphragmatic approach is being used. The tube should be no smaller than a No. 18 French. The presence of blood in the gastric contents is of no aid in assuming involvement of the stomach, for the patient usually swallows the blood he does not cough up. In perforating wounds with one of the wounds in the region of the kidney or in penetrating wounds with the missile in the flank, the urine should be examined for the presence of blood.

In the patients who do not respond adequately to resuscitation measures and in whom the chest involvement is felt to be under control, the factors of continued intraperitoneal hemorrhage and/or continued intraperitoneal contamination from large bowel contents should be kept in mind. Gross contamination of the left pleural cavity by contents of the large bowel will result in as profound a depth of shock as is seen. These patients will respond, if at all, only after the source of the contamination has been removed. Early surgical intervention is necessary when the right lobe of the liver continues to bleed into the pleural cavity or when the spleen lies shattered in the left pleural cavity. One must be able, on the basis of the position of the wound of entrance and the position of the fragment in relation to the lack of response to shock therapy, to evaluate the probable situation and proceed with surgery without further delay in spite of the hypotension. The return of a more normal blood pressure after clamping the splenic vessels or packing the liver is most gratifying. But its return in fecal contamination of the peritoneum and/or pleura is not so prompt and may not occur if the contamination has been severe and of long duration. There is also an occasional case in which the airway becomes the primary problem because of excessive bleeding into the tracheobronchial tree. If bleeding continues after a thorough endotracheal catheter suction, further delay is unwise, and the patient is removed immediately to the operating tent for rapid induction and endotracheal intubation which permits a more complete tracheobronchial toilet to be done. In our experience this method of management of the latter group of patients has so improved their general condition that the necessary surgery could be accomplished without further delay.

PENETRATING AND PERFORATING WOUNDS

Treatment of penetrating and perforating wounds is, in the main, based on the presence or absence of continued intrathoracic hemorrhage. Thoracotomy is necessary to control those which are bleeding, whereas those not bleeding are treated according to the anatomic severity of the injury. The latter are treated by local débridement and closure of deep muscle layers. Sucking wounds require enlargement for proper closure. Wounds causing large chest wall defects will be discussed under Traumatic Thoracotomy.

The diagnosis of continuing intrathoracic hemorrhage is the most difficult diagnostic problem in thoracic surgery in forward installations. It has been pointed out that death from hemorrhage from the pulmonary parenchyma is unlikely. We saw four instances of bleeding of considerable magnitude from lacerations of the upper half of the upper lobes which required suturing. There was no similar degree of hemorrhage from lacerations of the same size in the remaining lobes. The reason may be that lacerations in this region involve vessels of greater size than comparable lacerations in other lobes. There remain the following sources of hemorrhage: intercostal (10 cases), internal mammary (2 cases), and mediastinal vessels (2 cases—aorta, hemiazygos, and accessory hemiazygos veins), and the heart (2 cases). These illustrate the fact that intrathoracic hemorrhage from sources other than intercostal vessels is relatively uncommon. Originally, we believed that intercostal vessels in the wounds should be visualized in the majority of cases. A record was kept of all intercostal arteries which were bleeding at the time of exposure and those which were transected or lacerated but not bleeding. In 200 cases, an intercostal artery was found bleeding only 10 times (5.0 per cent), whereas it was found transected or lacerated but not bleeding 33 times (16.5 per cent). All bleeding vessels were found in one of two areas: first, in the area from the origin of the vessel to the angle of the rib and, second, in an area extending approximately 6 cm. from the lateral border of the sternum. In view of these findings, it was felt that more conservative treatment could be practiced. In the latter part of the series, 35 penetrating and perforating wounds were locally débrided without investigating the continuity of the intercostal vessels. There were no deaths in this group. It is reasonable to assume that some of these patients had involved but nonbleeding intercostal vessels. This makes the proportion of nonbleeding to bleeding intercostal arteries more marked.

The knowledge of the percentage chance of the presence or absence of a bleeding intercostal artery is not of great help in the individual case. Each case must be carefully evaluated on the basis of individual findings. The presence of a large hemothorax is not an indication for an exploratory thoracotomy. Aspiration can be done and a check roentgenogram and reaspiration will permit the evaluation of the patient's status. This program works well during periods of relative quiet when only a few patients are admitted. During a "push," however, the surgeon is presented with a large number of patients, and his judgment will often be sorely tried. During the period of observation of patients with suspected bleeding, the traumatic thoracotomies and thoracoabdominal wounds can be treated. To attempt a diagnosis of continued intrathoracic

bleeding solely on the basis of changes in pulse rate, blood pressure, and respiration will not infrequently lead to error, for some patients remain in relatively good condition with actively bleeding intercostal vessels. Judgment of the problem cases becomes more accurate in proportion to the number of cases seen. No deaths from bleeding vessels have occurred in this series. However, from the number of involved but nonbleeding intercostal arteries which we have uncovered, we feel that it is better to investigate a case if any doubt exists. One should keep in mind the possibility of the postoperative recurrence of intercostal hemorrhage in the patients whose intercostal vessels were not exposed. It did not occur in this series, but we have been informed of two fatalities from hemorrhage on the second and fourth postoperative days.

In wounds which were not sucking and were not the source of bleeding, débridement of the chest wall muscle was done avoiding, when possible, the opening into the pleural cavity. Endotracheal anesthesia was used, because occasionally a simple wound started sucking during débridement. After débridement, these wounds should have at least a two-layer closure of the chest wall muscle. Skin and subcutaneous tissue were left unsutured.

Sucking wounds necessitated an exposure adequate for a proper closure. With the wound in the intercostal space, the pleura had a defect best closed by including a deep layer of the chest wall muscle in the line of sutures which included the intercostal muscle. At least two more muscle layers were usually available for closure with interrupted silk sutures. In the anterior portion of the lower costal margin, imbrication of the single muscle layer of the chest wall was desirable in order that more than one muscle layer, besides the intercostal one, be used in closure. When the wound involved a rib, the fragments dangling into the pleural cavity were removed. If the intercostal nerve was readily found, it was crushed to relieve postoperative pain. The continuity of the intercostal vessels was checked at this time. The skin and subcutaneous tissue were left unsutured.

The problem of removing foreign bodies from the lung or pleural cavity should be considered in the light that in the majority of patients they are tolerated without endangering life. Removal of a foreign body is not of itself sufficient indication for thoracotomy in the forward areas. The exceptions to this generalization are the patients in whom the foreign bodies are in potentially dangerous positions, such as in the hilus of the lung or in the mediastinum in the region of the esophagus. However, during closure of a sucking wound, exploration for source of continued bleeding, or exploration to investigate diaphragmatic involvement, any readily available foreign body should be removed. The position of the foreign body was recorded in 75 of the 84 penetrating wounds. Forty-one (54.7 per cent) were found in the lung, 18 (24.0 per cent) in the pleural cavity, and 16 (21.3 per cent) in the chest wall; the last traversed the lung. In twelve patients, the foreign bodies were readily available in the lung during surgery for sucking wounds and were removed. In two instances, thoracotomy was done because of the presence of missiles. In both cases, they were very large and in the hilus of a lobe. Foreign bodies were removed from the pleural cavity in thirteen patients. In five, thoracotomy was

performed because it was thought that the missile had torn the right diaphragm and penetrated the liver. With a pleural cavity incompletely clear of blood, it is often difficult to ascertain the position of the foreign body in relation to the diaphragm. The exact position of a fragment deep in the costophrenic sulcus may be misleading. The other foreign bodies were readily available and were removed from the pleural cavity during débridement. It is suspected that metallic bodies, regardless of size, are tolerated in the pleural cavity better than we originally thought. It may well be that thoracotomy for this purpose in the forward areas is unnecessary and that it can be delayed until the patient arrives at the chest center. In these and in all patients with fragments remaining in the lung, all efforts should be directed toward rapid and complete expansion of the lung.

Earlier we felt that when exposed, lacerations of the lung should be sutured. But this lacerated tissue is the site of interstitial hemorrhage, and the question arose as to whether or not the suture would hold long enough to serve its purpose. This suspicion was furthered, to some extent, by the postoperative pleural pressure readings. Recently, many lacerations were left unsutured and no untoward effects were noted. The lungs expanded as well and in the same time as those that were sutured. This again bears out the idea that the surrounding airless lung, infiltrated by blood, reduces the occurrence of postoperative parenchymal-pleural fistula. At present, suturing lacerations of the lung is reserved for the following: obvious parenchymal-pleural fistulas, very large lacerations (to reduce the amount of postoperative drainage), torn parenchyma that is bleeding into the pleural cavity, and lacerations which are the source of bleeding into the tracheobronchial tree. The suture of the last is particularly helpful when it is difficult to keep the tracheobronchial tree free of blood preoperatively and when repeated endotracheal suction is required during the operation.

TRAUMATIC THORACOTOMY

The term traumatic thoracotomy is arbitrarily reserved for excavated wounds, palm-sized or larger, involving the pleural cavity. There were six of these in the series. This term also includes those perforating wounds which, after connecting the wounds of entrance and exit by mere skin incision, reveal a similar defect. There were five of this type. There were no deaths in these eleven cases. The traumatic thoracotomies occurred chiefly in the anterolateral portion of the lower costal margin. On the right side, the diaphragm and liver were lacerated and, on the left side, the diaphragm, spleen, and occasionally the kidney were involved. The management of these associated injuries will be discussed under Thoracoabdominal Injuries.

The treatment of traumatic thoracotomy requires methods which eliminate paradoxical motion of the chest wall and permit closure without tension. As there are always numerous rib fractures, the wiring of a few will eliminate paradoxical motion. The use of muscle flaps is of great help in closing large defects without tension. In our series, the largest wound of this type occurred on the left side. The wound of entrance was just to the left of the spine of the

fourth dorsal vertebra and the wound of exit was in the left flank. The skin incision connecting the two wounds revealed an entire "blowout" of all the chest wall and intercostal muscles with fractures of the fifth to twelfth ribs, inclusive, just lateral to the transverse processes. In débriding such a wound one must be radical, for there is often gross contamination of the muscles of the chest wall by dirt and clothing. Large masses of muscles are commonly so badly contused as to be nonviable. Débridement will further enlarge the defect. During this procedure, it is not necessary to remove rib ends merely because an irregular fracture is present. Fractured ribs will be of aid in the closure. If more than four ribs are fractured, two or three of the centrally placed fractures should be wired. Wiring is done by inserting fine steel wire into the small holes drilled in the ends of the fractured ribs. In one patient with a large traumatic thoracotomy with fractures of the fifth to twelfth ribs, marked paradoxical motion was evident preoperatively. The result of wiring the seventh, eighth, ninth, and tenth ribs was the almost complete absence of paradoxical motion postoperatively. The patient's postoperative course was uneventful, and he was evacuated with a fully expanded lung. Comminuted fractures with defects in the ribs present a difficult problem. When there are not more than two, wiring of the fractured ribs above and below them will suffice. In patients with as many as five or six fractured ribs with defects, the ends of at least the two centrally placed ribs should be wired even though their ends cannot be approximated.

Nonabsorbable material is used to permit fixation of the chest wall to occur because it will hold during the first ten-day critical period when a strenuous postoperative cough routine is essential. It is believed that absorbable material will not hold sufficiently long under this regimen. Nonabsorbable material is used in spite of the fact that in such contaminated wounds some will probably develop osteomyelitis of the ribs at a later date. In view of the patient's poor condition before surgery, methods which give immediate excellent results must bear more weight than the consideration of the possible development of later complications.

Undermining and mobilizing adjacent muscles is helpful but usually is insufficient to close the defect without tension. If there is any tension, even a small amount of infection will rapidly result in an infected sucking wound with a total empyema. Muscle flaps should be swung into place. It has been found in planning the flap that its length should be three-fourths greater than the distance from its base to the far side of the defect. It can then be sutured in position without tension because allowance is made for the retraction which occurs when the flap is cut. To obtain the desired three-layer closure, the deep fascia of the flap is used as the first layer, and the other layers are formed by imbricating the split edge of the flap and the muscles adjacent to the defect.

THORACOABDOMINAL WOUNDS

A thoracoabdominal wound is defined as one caused by a single missile which involves the pleural cavity, diaphragm, and peritoneal cavity. There were 55 thoracoabdominal wounds in this series. Of these, 36 were right sided, 16 left

sided, and 3 bilateral. In the last, a single missile involved both pleural cavities and both diaphragms. There were 9 deaths (16.4 per cent). This mortality is uncorrected for the factor of concomitant wounds. Intra-abdominal organs were involved as shown in Table I.

TABLE I. INVOLVEMENT OF INTRA-ABDOMINAL ORGANS

ORGAN	NUMBER	PER CENT
Liver	38	69.1
Spleen	12	21.8
Stomach	10	18.1
Large bowel	7	12.7
Kidney	6	10.9
Small bowel	3	5.4
Pancreas	2	3.6
Gall bladder	1	1.8
Common duct	1	1.8

The mortality is greater with left-sided wounds in which massive contamination of the pleural cavity and/or peritoneal cavity from contents of the large bowel occurs. Of the 19 combined left-sided and bilateral wounds, there were 7 deaths (36.8 per cent). Three of these involved the splenic flexure of the large bowel. The causes of death in the other 4 were: cerebral hemorrhage (embolism?); abscess in the region of a nephrectomy; and (bilateral case) massive hemorrhage from the liver in which both lobes were broken across in their long axis. Of the 36 right-sided wounds there were 2 deaths (5.5 per cent), one from hemoglobinuric nephrosis, and the other from bile peritonitis resulting from an undiscovered nick in the common duct at its junction with the pancreatic duct. There were 3 deaths (42.9 per cent) in the 7 patients with large bowel involvement. A similar experience was noted by Betts.[2] In his total of 29 thoracoabdominal wounds, there were 6 deaths (20.7 per cent). In his series there were 5 deaths in the left-sided wounds (27.7 per cent), and the large bowel was involved 8 times with 3 deaths (37.5 per cent). These mortalities are more than acceptable in view of Hurt's findings of 135 deaths (47.2 per cent) in the follow-up of 286 patients with large bowel injuries.[3]

The position of the foreign body in a penetrating thoracoabdominal wound must be accurately located. Anteroposterior and true lateral roentgenograms of the abdomen are essential. Careful examination of the wound will occasionally be quite revealing. For example, the odor of *Bacillus coli* in two left-sided wounds was preoperative evidence of large bowel involvement, and the probability of gross pleural contamination indicated early surgery. Omentum was seen in three wounds and pericardial fat in one. It is likely that the omentum and large bowel were sucked up through the torn diaphragm by respiratory action whereas the pericardial fat was apparently driven into the wound of exit. These findings may be helpful in establishing the direction of the missile.

Left-Sided Perforating Wounds.—Perforating wounds in the lower costal margin should always be examined and treated for the possibility of diaphragmatic involvement. If the wounding occurs during the expiratory phase, the anatomic position of the wounds in the chest wall may be quite cephalad and yet there may be a torn diaphragm and injured peritoneal organs. In one instance of laceration of the diaphragm and spleen, the wound of entrance was in

the fourth interspace just medial to the left nipple and the wound of exit was in the seventh interspace in the left midaxillary line. Perforating wounds of this region should be prepared as expected thoracoabdominal wounds. It is a simple matter, when débriding these wounds, to enlarge the one nearest the diaphragm to permit examination of this muscle. The chest wall is relatively thin in this region and, consequently, the majority of wounds are sucking. In the process of closure, enlarging these wounds will permit an adequate check on the diaphragm. Involvement of the left diaphragm is of itself sufficient indication for thoracotomy. To reduce postoperative wound infection in diaphragmatic involvement, it is best, if anatomically feasible for good exposure, to resect a rib which is not in the wound of entrance or exit.

Left-Sided Penetrating Wounds.—Accurate localization of the missile, whether or not it is in the upper abdominal quadrant, is extremely important in left-sided penetrating wounds because its location determines the type of approach. Any damage done to intraperitoneal organs by a missile which enters the left upper quadrant and remains there can readily be reached and remedied by a transdiaphragmatic approach. This approach permits access to this region with greater technical ease than is attained through a laparotomy approach and the usual postoperative complications are decreased. However, if the missile passes into any other quadrant of the abdomen it is safer to perform a laparotomy.

The left transdiaphragmatic approach has many technical advantages which permit the following to be done with ease: splenectomy; exploration and repair of the entire stomach from the esophagogastric junction to the duodenum; examination and mobilization of the colon from the hepatic flexure to the beginning of the sigmoid loop; subcostal or midline exteriorization of the colon by either loop or spur colostomy; inspection and treatment by suture or resection and anastomosis of the small bowel from the ligament of Treitz to within a few inches of the cecum; and visualization and removal of the left kidney.

After anesthetization, the patient is placed on his right side with a folded blanket under the lower lateral portion of the right costal margin. A long strip of adhesive placed over the left hip and attached to the litter anteriorly and posteriorly fixes the patient in position. The left leg is flexed at the hip and knee with a pillow beneath the knee. With the right leg extended, the great saphenous vein anterior to the medial malleolus or at the medial border of the middle of the tibia is available for the administration of fluids during the operation. Removal of the ninth rib usually gives the best approach to the left upper quadrant. However, as this varies with habitus of the individual, the proper rib can be selected during débridement of the penetrating wound. The rib resection should stop short of the costal cartilage to prevent the occurrence of a chondritis if contamination is present. In opening the diaphragm, the wound of penetration should be included, if possible. When a splenectomy is indicated, the lateral peritoneal reflection over the spleen is easily cut, and the spleen is delivered into the opening in the diaphragm. The splenectomy is technically easier if, after delivery, the operator and the assistant change sides. The assistant is behind the patient and maintains the delivery of the spleen with his left hand. The operator, facing the patient, can then pass his fingers be-

neath the splenic pedicle, and this decreases the possibility of injury to the greater curvature of the stomach and the tail of the pancreas. In exploration of the stomach, opening of the gastrocolic omentum permits access to its posterior wall. For exposure of injuries in the region of the esophagogastric junction, especially for those on the lesser curvature side, it is necessary to cut the left triangular ligament and mobilize the left lobe of the liver. To expose the left kidney, the lateral peritoneal reflexion of the upper part of the descending colon and the lienocolic ligaments must be cut, and the colon laid mesially. This approach permits a nephrectomy to be done easily.

Two layers of interrupted silk are used to close the diaphragm. It is important that each suture of the first layer should include the peritoneum. There is no need to crush the phrenic nerve. The apparent defect in the lateral costal margin of the diaphragm is a result of contraction after severance of the attachments. It is repaired by suturing the diaphragm to an intercostal muscle one or more spaces higher than its original attachment. This principle of reattachment is important, for the original site is usually the area of the wound of the chest wall with its accompanying fractures and will not be stable enough for efficient diaphragmatic action. When the diaphragmatic crura are torn from the vertebral column, the first layer of sutures should be passed through the superior surface of the lobe of the liver. A sufficient number of the sutures will hold, fixing the liver against the undersurface of the diaphragm and preventing herniation.

Occasionally a left thoracoabdominal wound of the penetrating type occurs without hemo- and/or pneumothorax. The foreign body is in the left upper quadrant. The wound of entrance is not sucking because of its oblique course through the chest wall. The question arises: with the possibility of a large bowel injury, should the approach be through the pleural cavity in which there is no preoperative evidence of contamination? We have not settled this conclusively because the problem occurred in only two of our cases, and both were handled through a laparotomy with closure of the diaphragm from below. However, considering the technical ease of doing these operations transdiaphragmatically as compared to doing them through a laparotomy incision, we propose in the future to do them transdiaphragmatically. The factors of postoperative collapsed lung and pleural infection need not figure prominently in the decision if proper emphasis is placed on the toilet of the pleura and on pulmonary expansion during closure of the thoracotomy.

Right-Sided Perforating Wounds.—Right-sided perforating wounds involve the lower third of the chest and are usually at a right angle to the ribs. Careful alignment of the wounds of entrance and exit will assist in assuming which organs are involved. In our series perforating wounds of this region involved only the liver and diaphragm. These may be divided into two types according to position in relation to the diaphragm and to size: the "blowout" type (traumatic thoracotomy) through which the diaphragm and liver are readily exposed, and the small wound at a distance from the diaphragm and liver which requires the resection of a rib not involved in the wound to give the desired exposure. In the latter type, the rib to be resected can be selected through the débrided wound.

It is felt that right diaphragmatic lacerations should be closed and the infradiaphragmatic space should be drained subcostally. The diaphragmatic injuries are always associated with a liver torn to some extent. If the diaphragmatic rent is not repaired, a biliary fistula into the pleural cavity may occur. Biliary pleural fistulas are in themselves difficult to treat and there is probably more than a casual relationship between biliary-pleural fistulas and pleural empyemas. This is particularly true if the laceration of the diaphragm is over the bare area of the liver. This region does not have available peritoneum to include in the closure of the diaphragm. The pooling of undrained blood and necrotic material from a liver tear can more readily break through a sutured diaphragmatic rent unfortified by peritoneum. In the patients in whom the missile lacerates the diaphragm and the liver in the bare area region, drains can be laid over the rent in the liver, and, by breaking through the lateral reflection of the peritoneum over the right lobe of the liver, the drains can be exteriorized subcostally.

Right-Sided Penetrating Wounds.—Twenty-six of the 36 right-sided wounds in the series were of the penetrating type. Such cases pose the problem of determining whether the foreign body has passed entirely through the right lobe of the liver and is in the peritoneal cavity, or whether it has stopped in the liver. When it is evident by roentgenogram that the fragment has stopped in the liver, the management is as just detailed in right-sided perforating wounds. The problem is then merely one of drainage of the suprahepatic space and repair of the diaphragm. If the fragment is readily available in the liver, it is removed. Protracted exploration for a buried foreign body will often result in considerable hemorrhage.

When the missile is revealed to be in the peritoneal cavity, the abdomen is best explored through a laparotomy approach. Wounds of the diaphragm no higher than approximately the eighth interspace and no farther back than the posterior axillary line can be exposed and sutured from below. The condition of the patient will determine whether or not the diaphragm, penetrated in other areas, will be repaired. It is obvious that a fatality will not occur from an unrepaired right diaphragm. The likelihood of a biliary-pleural fistula occurring cannot be considered of major importance in the patient who is in poor condition because of intraperitoneal injury and in whom a laparotomy is necessary. In such instances, débridement, closure of the chest wound, insertion of an intercostal tube, and aspiration of all blood and air precede the laparotomy. These are done to establish and to maintain as high a vital capacity as possible during the laparotomy. Otherwise, blood from the liver draining into the pleural cavity may result in a considerably lowered vital capacity by the time the laparotomy is completed. The intercostal tube drains into a water-trap bottle and indicates the amount of liver bleeding. This procedure was necessary in only two of our patients, one of whom developed a biliary-pleural fistula. When the patient's physical status permits, a thoracotomy with repair of the diaphragm and expansion of the lung precedes the laparotomy and suprahepatic drainage. This amount of surgery was performed in the majority of cases in our series and was not prohibitive, but it is to be applied only after careful evaluation in each case.

INJURIES TO MEDIASTINAL STRUCTURES

Exploration for injury to mediastinal structures is done for one of two reasons: suspected injury to the esophagus, and continued bleeding from large vessels or from the heart. A small fragment whose position and course is such that neither of these is suspected need not be removed. Enlargement of the mediastinum by extravasation of blood as seen by roentgenogram does not merit exploration unless it is accompanied by signs of progressive swelling of the neck veins. We have learned that neck wounds also may present a markedly enlarged superior mediastinum. The enlargement can be caused by blood from a neck vessel dissecting into the mediastinum. Before proceeding with such extensive exposures as removal of a part of a clavicle and a portion of the sternum, it is well to investigate thoroughly the neck wound because the source of hemorrhage may be reached from above. The source of hemorrhage may be available by exposure in the base of the neck even when the missile is in the superior mediastinum. With the missile in this region not easily removed, the superior mediastinum can be drained through the cervical wound of exploration.

Diagnosis of injury to the thoracic esophagus is very difficult. Dysphagia was not noted in our series. However, severe back pain radiating down into the lumbar region was a constant complaint and was unrelieved by a large dose of morphine. There have been no constant pathognomonic roentgenographic findings. Air in the mediastinum was seen only once. The only way that mortality from esophageal injury could be reduced would be by doing a thoracotomy on every suspected injury. Accurate preoperative diagnosis is far from the rule. The picture is always obscured by the hemopneumothorax hiding the missile's unpredictable path in traversing the lung. However, keeping in mind the exact position of the esophagus in the various portions of the thorax in relation to the position of the missile as determined by accurate anteroposterior and lateral roentgenograms will be of some help. But even this help is not available when the missile passes through the mediastinum.

The basic principles of treating lacerations of the thoracic esophagus are repair of continuity, decompression by posterior paravertebral mediastinotomy, and diversion of subsequent infection from the pleural cavity by repair of the mediastinal pleura. The esophageal repair is often difficult, and some may leak even when well repaired. In others, the position of the laceration, such as at the level of the arch of the aorta, prevents adequate exposure and may be left unsutured. All these possibilities are covered by establishing a mediastinotomy and closing the rent in the mediastinal pleura. The approach should be a posterolateral transpleural one as close to the level of the suspected injury as possible rather than that of a posterior mediastinotomy, for the latter is too limited in its exposure for associated hilar and mediastinal injuries. The esophagus, especially the upper third, is best approached from the right side, but, when the wound of entrance and the missile are both on the left, no choice is afforded. Closure of the esophagus is done in its transverse axis and should consist of two layers. A continuous fine silk suture is used for the inner layer and interrupted silk mattress sutures for the outer ones. Care should be used to make certain that the two layers are distinct in that the ends of the inner

layer of sutures do not protrude into or through the outer layer. If the Levine tube cannot be passed preoperatively, it may be introduced and guided past the injury during the repair. The mediastinotomy is performed during the thoracotomy in the following manner: the mediastinal pleura is dissected for a considerable distance in all directions; a three-inch needle is slid alongside the vertebral body in the mediastinum at the level of the injury—the needle point is pushed out of the skin posteriorly and the hub of the needle is left in the intercostal space at the level of the esophageal rent; a Penrose drain is placed alongside the laceration and packed down to the intercostal space alongside the body of the vertebra; the mediastinal pleura is then closed. When a pleural defect makes closure difficult, a flap is raised from the parietal pleura and the closure is accomplished without tension. The remainder of the posterior paravertebral mediastinotomy is done using the needle as a guide to remain extrapleural and to lead directly into the mediastinum to the drain. Segments of ribs, transverse processes, and intercostal muscle bundles are resected above and below the needle. The end of the drain is exteriorized. It is well to remember that mediastinotomy wounds close rapidly. Therefore, gauze should be inserted to keep the wound edges and the intercostal space open.

We found two types of hemorrhage from the cardiac chambers: active and "controlled." An example of active hemorrhage was found in a patient in whom no tamponade existed because of decompression by laceration of the pericardium. A picture was presented of massive hemothorax in which continuing hemorrhage was suspected. At operation, a widely lacerated pericardium was found, and a laceration of the myocardium was discovered as the source of hemorrhage. "Controlled" hemorrhage was typified by a case in which the missile entered the posterolateral aspect of the junction of the right auricle and the superior vena cava. The pressure of the resulting tamponade proceeded only to the point of cessation of the hemorrhage. Opening the pericardium relieved the tamponade, and the hemorrhage did not recur. Both patients made an uneventful recovery. "Controlled" hemorrhage raises the problem of the use of pericardial aspiration to replace operative treatment. It is of aid in preoperative management, but in tamponade caused by irregular fragments it would be hazardous to depend on this type of treatment alone in all such cases. Repeated aspiration may be sufficient in some patients but it would appear to be safer to operate on any patient in whom tamponade reappears after two aspirations.

We had two patients with hemorrhage from large mediastinal vessels. One had lacerations of the hemiazygos and accessory hemiazygos veins and the other had two small holes in the thoracic aorta. Surgical intervention in the latter was quite unsuccessful.

CHEST WALL WOUNDS

Chest wall wounds without penetration of the pleura seldom present major problems. In 64 chest wall wounds, we found 6 (9.4 per cent) instances of associated pneumothorax. These pneumothoraces responded to simple thoracentesis. There were 12 (18.8 per cent) instances of contusion of the lung underlying the chest wall wound. These hemorrhagic contusions all cleared without sequelae in nine to twelve days. Considering this incidence of pneumothorax and con-

tusions of the lung, it is suggested that roentgenograms be made for all chest wall wounds.

Another problem that occasionally occurs is the involvement of the subscapular space. There were 6 (9.4 per cent) such lesions in our series. In wounds traversing the subscapular space, the possibility of anaerobic gas infection should be kept in mind. To do a complete débridement, an incision approaching the magnitude necessary for a thoracoplasty and mobilization of the scapula would be necessary. This was not done in these cases. Instead, incisions were made along the track of the missile at the vertebral and axillary borders of the scapula, and injured muscle and foreign material were removed. Through-and-through Penrose drains were inserted. A dependent drain was placed in the subscapular space through an incision in the triangle of auscultation at the inferior angle of the scapula. No gas infection developed in these patients. All such patients should be kept at least 6 days postoperatively. At any rise in pulse rate, occurrence of pain, or change in the patient's demeanor, the wound should be examined immediately. Missiles remaining in the subscapular space should be removed and thorough drainage instituted. These precautions should eliminate or at least reduce the incidences of subscapular abscesses. Elimination of the possibility of these abscesses is important, for they may go undetected for long periods.

COMBINED THORACIC WOUNDS AND SPINAL CORD INJURIES

In this series, four patients with chest wounds had associated spinal cord injuries. One had a penetrating wound of the chest in which the missile was pressing on the cord. Three had perforating wounds with a traumatized spinal cord. In débridement and exposure to close the adjacent sucking wounds, removal of fragments of the laminae disclosed the extent of the cord injury. In two, the cord was markedly swollen but intact; in one, the cord was the site of extensive hemorrhage; and in the last, the cord was transected. All cases presented the picture of transverse myelitis.

The mortality was in direct relation to the skin anesthesia level. The two patients who died had skin anesthesia above the umbilicus (one just below the nipples and the other at the xiphoid). The two who lived had skin anesthesia levels below the umbilicus (one just below the umbilicus and one halfway between the umbilicus and symphysis pubis). All four patients had approximately the same amount of lung and pleural damage. The two deaths occurred four or five days following operation from pulmonary edema that did not respond to treatment. Positive pressure oxygen therapy and endotracheal suction were of no avail. Apparently, paralysis of the abdominal rectus muscles reduces the efficiency of the cough mechanism and may be a factor in permitting pulmonary edema to ensue in the acutely injured. The tone of the recti in a gentle cough or actual recti contraction during a vigorous cough gives the diaphragm an opposing intra-abdominal pressure. When the opposing pressure is lost by paralysis of the recti, the cough becomes ineffective because the expulsive action merely bulges out the abdomen. Possibly a snug abdominal binder or a plaster abdominal jacket for the first four or five days may be efficacious in replacing the function of the recti. With the innervation of only the upper half of the recti

intact, the cough mechanism can function efficiently. From these few cases, it seems that a patient with a penetrating or perforating injury to the lung and injury to the spinal cord has a poor prognosis if the skin anesthesia level is above the umbilicus.

PRINCIPLES OF PULMONARY EXPANSION

Originally, drainage was carried out intercostally in any patient with lacerations of the lung, contamination of the pleural cavity, or abrasions of the parietal pleura. The intercostal tubes usually functioned for only two days. Aspiration was then necessary. Recently, following the suggestion of Betts and Lees,[2, 4] thorough pleural lavage was done in addition to expanding the lung completely at the time of operation, thus decreasing the use of intercostal drainage. Lung expansion has been quite satisfactory and thoracentesis has been done less frequently. The recent change from drainage to nondrainage is not as important as the factors of *thorough pleural lavage and complete expansion of the lung at the time of closure*. If these two procedures are carried out conscientiously obliteration of the pleural space has a good start. Any pleural fluid which then forms is of a serous type that lends itself to aspiration more easily than does a grossly bloody fluid containing clots. Adherence to these principles may eliminate total empyema and should reduce the percentage of organizing fibrothorax. We do not recommend that the generally accepted principle of intercostal drainage in military surgery be revised but in light of our experience during the past few months we feel its application in such cases need not be practiced routinely. Whether or not a drain is used is not as important as proper toilet of the pleura and complete expansion of the lung at the time of operation.

Frequently, during expansion of the lung by the anesthetist, we have noted that the lateral portion of the lower lobe did not expand as readily as the remainder of the lung. With the patient on his side, the partial collapse of the lung permits a folding over of this portion onto the diaphragmatic surface of the lower lobe. When it is pulled out and unfolded, aeration occurs.

POSTOPERATIVE CARE

Postoperative care should focus on the main objective of complete and early pulmonary expansion. In this respect, the operative and postoperative period should be considered as one. The goal, a completely expanded lung, is achieved at the time of closure of the wound, and is maintained by repeated thoracentesis when even small amounts of pleural air and/or fluid are present.

Maintenance of a clear tracheobronchial tree by an energetic cough routine is aided by intercostal nerve block when pain is an inhibitory factor, and is another step in maintaining aeration of the lung. When the airway is not cleared by these methods we resort to tracheal aspiration. Postoperative bronchoscopy is necessary and should not be delayed if these measures have failed. Sedation is kept at a minimum to insure prompt cooperation with the cough routine. Nasal oxygen is given for postoperative shock and is continued only in those patients in whom pulmonary moisture persists. When the need for oxygen continues, one may be certain that all of the available parenchyma is not being

utilized and a check should be made for pneumothorax, for increasing pleural fluid, for inhibition of the depth of respiration by pain, or for occlusion of a bronchus by secretions.

ANESTHESIA

The preparation of our patients with intrathoracic injuries was begun in the resuscitation tent. In carrying out the procedures already described in the preoperative section, the anesthetist is better prepared to evaluate the patency of the airway, the physical status of the patient, and the need for premedication; and to select the agent and technique to be employed.

For certain absorption and for optimum effects at the time desired, premedication was nearly always administered intravenously in the operating tent. Atropine, gr. $\frac{1}{100}$, was given to all patients except those in shock with an extremely rapid pulse. Morphine, in doses of gr. $\frac{1}{12}$ to $\frac{1}{4}$, was given to all who needed sedation. Not infrequently, acute morphine intoxication was observed when the circulation improved in response to resuscitative measures and the morphine given enroute to the patients in shock was rapidly absorbed.

The inducing agents were pentothal sodium, nitrous oxide and oxygen, and ether. Pentothal was used in 53.0 per cent of the cases. This drug was employed so frequently because its quick action made possible the rapid induction desired in patients with continuing bleeding into the tracheobronchial tree. It was the agent of choice for the maintenance of the anesthesia for some of the patients, and of necessity during the Sicilian and early part of the Italian campaigns when neither nitrous oxide nor anesthesia machines were always available. For that period, an apparatus was improvised to administer oxygen and to expand the lung. Oxygen is administered before, and continued during, pentothal induction. Before starting this regimen, we saw several severe laryngospasms associated with marked cyanosis when the laryngoscope was inserted. Ether was used for induction in 9.5 per cent of the cases. The seriously wounded patients with no obstruction were induced with ether alone without difficulty. At the present time, nitrous oxide and oxygen are employed to induce all except those who require rapid induction and rapid endotracheal intubation.

Inhalation agents are poorly absorbed when the airway is constantly refilling and, as a result of this, inductions with them are prolonged and difficult. With experience, intubation may be done easily, rapidly, and early in the induction with pentothal, and after the airway is cleared, anesthesia with inhalation agents may be begun. It is inadvisable for an unskilled anesthetist to use an uncontrollable agent, such as pentothal, in the presence of a partially obstructed airway.

Ether was used for maintenance in 85.0 per cent of our patients and pentothal in the remainder. Ether was perfectly satisfactory in every instance. To determine its merits, pentothal was employed in twenty-two consecutive severely wounded patients requiring major thoracotomy. Its advantages are that controlled respiration is more easily effected, respiratory movements interfere minimally with the surgical procedure, and no irritation of the bronchial mucosa is produced. In winter fighting, many of the wounded had severe

bronchitis. Good results were obtained because minimal doses were used and this was possible because deep anesthesia was unnecessary in these operations not requiring relaxation. Pentothal has the disadvantages of greatly reducing respiratory exchange and depressing the patients for longer periods postoperatively than is desired. A cough routine should be started soon after completion of the operation.

Ether, after a nitrous and oxygen or a pentothal and oxygen induction, is now employed in the majority of cases. Pentothal is no longer used as the principal agent because of its small margin of safety, but from our experience we feel that, in the hands of a skilled anesthetist, it may be used when an anesthesia machine is not available and improvisation is necessary.

In this series, all cases with wounds involving the pleura were handled with endotracheal and supplementary positive pressure anesthesia. The endotracheal technique facilitated the necessary frequent tracheal aspirations and the complete control of respiration without inflating the stomach. The respiratory exchange was increased by rhythmic manual compression of the breathing bag throughout the operation in many of the operations done with either and in all done with pentothal.

The physical status of many patients improved during prolonged operations. This was due to increased efficiency of respiratory exchange as a result of clearing the airway and using high oxygen concentrations with supplementary positive pressure; intravenous replacement therapy (blood); and a modified Trendelenburg position. Expansion of the lung was begun fifteen to twenty minutes before the closure of the pleural cavity. There were no deaths attributable to anesthesia.

It is a well-known fact that repeated aspirations of the trachea by means of a catheter passed through an endotracheal tube will efficiently clear the airway of blood, mucus, and fluid vomitus. To determine how efficient this technique is, bronchoscopy was routinely performed at the completion of fifty-four operations on patients who had gross blood in the tracheobronchial tree during the operation. We found that when the anesthetist believed that the tracheobronchial tree was completely cleared by catheter suction, no significant amount of blood was found by bronchoscopy. Therefore, at the present time, bronchoscopy is reserved for those patients with excessive tracheobronchial bleeding, and for the few who require examination for patency of the bronchi during the operation because too great a resistance is offered to pulmonary expansion.

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FOREIGN BODY, NEEDLE STYLET, IN THE PLEURAL CAVITY; REMOVAL THROUGH THORACOSCOPIC PROCEDURE

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MEDICAL literature contains numerous reports of foreign bodies located in the gastrointestinal tract, the bronchial tree, and the lower urinary system. These structures communicate with the exterior and thus allow for an easy and natural entry for foreign bodies. The pleural cavity, lacking such communication with the exterior, could be expected to become the site of such bodies only under exceptional circumstances. There are two methods whereby such a phenomenon can occur. A foreign substance can be aspirated into one of the bronchial branches. This is followed by an inflammation of the adjacent tissues into which the foreign substance bores and then, after penetrating the wall of the bronchus, passes into the pleural cavity. Such a case was reported by Davidsohn.[1]

His patient, 33 years old, was admitted to the hospital in February, 1939, with a diagnosis of subacute bacterial endocarditis. She died one month later. At autopsy, in addition to the findings of a characteristic subacute endocarditis, it was also noted that the left lung was adherent to the lateral chest wall. The diaphragmatic surface of the left lung was free of adhesions, but attached to its inferior surface was a small deciduous molar tooth. Except for the external adhesions, there was no evidence of old or recent pulmonary infection. The author commented that since deciduous teeth are usually not shed after the twelfth or thirteenth years, it must be assumed that the patient had aspirated this tooth some nineteen or twenty years before and that, after lodging in a bronchus, it had perforated its wall and had passed into the pleural cavity.

A somewhat similar case was reported by Carpenter.[2] His patient aspirated or swallowed four artificial teeth during a coughing spell. Some thirteen years later the patient developed pain in the chest and died after a long illness. At autopsy, four artificial teeth were found in the pleural cavity. A fistulous opening was noted in the external surface of the lung leading into an abscess cavity. There was no evidence of a scar in the esophagus. Poulet[3] reported several instances in which the pleural cavity was entered after the foreign body had perforated the wall of the esophagus.

The second method whereby such substances find their way into the pleural cavity is through the chest wall proper. This occurs most frequently after operations for empyema, etc., where the drainage tube or part of it slips into the pleural cavity. Besides this and the occasional accidental retention of gauze sponges, a new type of foreign body has been encountered recently, and

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because it may be met with more frequently in the future, its description will be of interest and may be of some value. Willauer and Chodoff[4] reported an instance in which a needle broke off at its hub while a pneumothorax treatment was being given. The needle lodged in the chest wall and, during an attempt to remove it through an incision in the chest wall, the needle was forced into the pleural cavity. A thoracoscopy was performed eight days later and the needle could be visualized partly imbedded in the chest wall. An alligator forceps was introduced through a second cannula and the foreign body was easily removed.

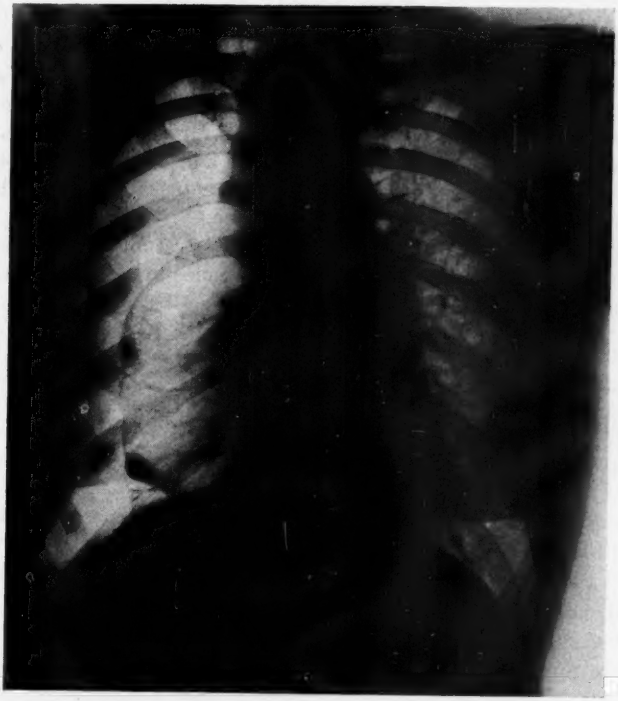

Fig. 1.—Roentgenogram taken two hours after accident, showing presence of the stylet free in part within the pleural cavity.

Andrews[5] reported a case in which a needle was broken during the aspiration of a pleural cavity. In this case also, an unsuccessful attempt was made to remove the broken portion of the needle by cutting into the chest wall at the site of the aspiration. Later that same day, through a thoracoscope, the needle could be seen protruding into the pleural cavity for three-fourths of its length and was readily removed through a second cannula.

The following report concerns a somewhat similar complication. While doing a routine pneumothorax refill, the resident physician noted that the stylet of his needle had slipped into the pleural cavity. A roentgenogram,

which was taken immediately after the accident, showed part of the stylet free in the pneumothorax space. In the early afternoon of that same day, Aug. 14, 1944, a thoracoscope was introduced into the ninth intercostal space at the anterior axillary line of the right side. The stylet could be readily seen running transversely from the chest wall toward the fissure between the middle and inferior lobes of the lung. As the lung moved during respiration, the stylet moved with it. On deep expiration, the two lobes could be seen to separate and it was then seen very clearly that the greater part of the stylet was not really embedded in the lung tissue but was lying free within the fissure. During inspiration and during quiet breathing, this part of the stylet was not visible because it was covered by the overlying lung. The free extremity of the foreign body was in contact with the chest wall but was not embedded within it, as could readily be determined by its free movement up and down with respiration. A second cannula was introduced on the same level in the line of the angle of the scapula. An alligator forceps was inserted through this cannula and, under direct vision, the free end of the stylet was grasped and without any difficulty extracted from the pleural cavity. The patient made an uneventful postoperative convalescence.

In view of the simplicity, effectiveness, and freedom from complications of this procedure, it should be utilized in preference to a cutting operation for the removal of suitable foreign bodies.

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